A CLINICAL STUDY ON MANAGEMENT OF

RETINAL VASCULAR OCCLUSIVE DISORDERS

Submitted in partial fulfillment for

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CERTIFICATE

This is to certify that the dissertation entitled, **"A CLINICAL STUDY ON MANAGEMENT OF RETINAL VASCULAR OCCLUSIVE DISORDERS"** submitted by **Dr. KALAIVANI. M,** in partial fulfillment for the award of the degree of Master of Surgery in Ophthalmology by The Tamil Nadu Dr.M.G.R.Medical University, Chennai is a bonafide record of the work done by her in the Regional Institute of Ophthalmology, Government Ophthalmic Hospital, Egmore, Chennai, during the academic year 2010-2013.

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RETINAL VEIN OCCLUSION

INTRODUCTION

Retinal vein occlusion is a retinal vascular disorder due to blockage of retinal vein either, the central retinal vein or any of the branch retinal veins, characterised by engorgement and dilatation of the retinal veins with secondary, mostly intraretinal haemorrhages and intra retinal edema, retinal ischemia including cotton wool spots, retinal exudates and macular edema and is usually associated with a variable degree of visual loss.

Retinal vein occlusion is a common cause of vision loss. It is the second most common cause of reduced vision due to retinal vascular disease, after diabetic retinopathy.

HISTORY

The dramatic picture of retinal vein occlusion was initially described as ‘retinal apoplexy’ by Liebreich in 1854. Leber in 1877 reported the first case of BRVO and called it ‘hemorrhagic retinitis’. It was first established as a clinical entity due to thrombosis by Julius von Michel in 1878. Koyanagi in 1928 first reported the association between BRVO and AV crossings.

INCIDENCE

The incidence of vein occlusion is 0.7% for the age group 49-60 years and 4.6% after the age of 80 years. It is currently estimated that there are about 520 new RVO cases per million people, of which 442 are BRVO and 80 are CRVO cases. [1] RVO typically occurs in middle aged and elderly individuals (older than 50 years), with an equal gender distribution.

ANATOMY

The four branch retinal veins, superior nasal and temporal and the inferior nasal and temporal veins, join to form the central retinal vein. Blood supply to specific retinal quadrant comes exclusively from the specific retinal artery and vein. Arterio-venous crossings occur more often in the upper temporal quadrant with the vein usually lying deeper than the artery at these crossings.

HISTOLOGY

The major branches of central retinal vein have thin walls made up of single layer of endothelial cells having a thin basement membrane, media is composed of elastic fibres, few muscle cells and a thin adventitia. The retinal artery and vein share a common adventitial coat.

PATHOPHYSIOLOGY

The exact pathogenesis is not understood fully. The clinical picture of CRVO may be explained by occlusion of main trunk of central retinal vein. Within the retrolaminar portion of optic nerve, the central retinal artery and vein are aligned parallel to each other in a common tissue sheath. They are compressed as they pass through rigid sleeve like openings in the lamina cribrosa. In addition, compression by an atherosclerotic central retinal artery may predispose to CRVO. The central retinal vein may also be subject to compression from mechanical stretching of lamina from increased IOP. Optic nerve head swelling and orbital disorders may also contribute to mechanical compression of the vein. Hemodynamic alterations such as diminished blood flow, increased blood viscosity and altered lumen wall (Virchow's triad) may lead to subsequent thrombus formation. CRVO is believed to result from thrombotic occlusion of the central retinal vein at or just posterior to the lamina cribrosa.

BRVO occurs at the AV crossing sites. At this site, the vessels have a common adventitial sheath. The artery lies anterior to the vein making the vein vulnerable to compression by the artery, resulting in turbulent flow, and predisposes to endothelial damage and thrombus formation. This is exacerbated by arteriosclerosis.

RISK FACTORS

Ocular risk factors

- Glaucoma
- Ischemic optic neuropathy
- Pseudotumour cerebri
- Tilted optic nerve heads
- Optic nerve head drusen
- **Systemic risk factors**
- Diabetes mellitus
- Hypertension
- Carotid insufficiency
- Hyperlipidemia
- Vasculitis
- Haematological disorders –

  Hyperviscosity syndromes **,**Lymphoma**,** leukemia,Anemia,elevated plasma homocysteine,factor XII deficiency,anti-phospholipid antibody syndrome, protein C protein, S deficiency, factor V Leiden mutation

CLASSIFICATION OF RETINAL VEIN OCCLUSION

CRVO – whole retinal venous system is involved

BRVO – involves only branches of retinal venous network

HRVO – if superior or inferior hemisphere of fundus is involved

CLASSIFICATION OF CRVO

1. Perfused/ Non ischemic / Partial

2. Non perfused/ Ischemic/ Complete/ Haemorrhagic

3. Indeterminate

CLASSIFICATION OF BRVO

1. Perfused/ Non ischemic

2. Non perfused/ Ischemic

Based on site of obstruction

Major BRVO

Macular BRVO

Peripheral BRVO

CRVO-CLINICAL FEATURES

SYMPTOMS

It can present with sudden painless loss of vision, transient obscuration of vision and visual field defects.

SIGNS

1. Visual acuity can range from 20/20 to even no PL in those with neovascular glaucoma. Visual acuity can be helpful in distinguishing perfused versus nonperfused CRVO.

2. Pupillary reaction - RAPD may be present in ischemic CRVO. It is measured by using neutral density filters.

3. Intra ocular pressure is elevated in case of glaucoma. Relative intraocular pressure difference is less helpful in the evaluation process. Immediately after CRVO, the IOP is typically lower in the affected eye as compared to the fellow eye. This relative difference diminishes with time and symmetry returns over the ensuing weeks to months.

4. Visual fields

Visual field testing is widely variable in CRVO. Abnormalities are more common and more severe in eyes with nonperfused rather than perfused CRVO.

5. Slit lamp examination may reveal iris new vessels.

7. Gonioscopy is essential to determine angle new vessels or angle closure from peripheral anterior synechiae.

8. Ophthalmoscopic examination

Typical features include superficial haemorrhages and deep blot haemorrhages in all four quadrants with dilated tortuous retinal venous system. The haemorrhages radiate from optic nerve head and are variable in quantity and may result in classic “blood and thunder” appearance. Optic nerve head swelling, cotton wool spots, splinter haemorrhages and macular edema are present to varying degree. Break through vitreous haemorrhage may be observed. An epiretinal membrane (ERM) may also develop. Optico-cilliary shunt can develop on optic nerve head, a sign of newly formed collateral channels with the choroidal circulation. Neovascularisation elsewhere (NVE) or neovascularisation at the disc (NVD) may develop as a response to secondary retinal ischemia. Fibrovascular proliferation from NVE/NVD may result in vitreous haemorrhage and tractional retinal detachment.

SEQUELAE

Vision

The Central Retinal Vein Occlusion Study (CVOS) [2] reported that eyes with initial visual acuity >20/40 or better have more favourable visual prognosis. Patients with vision worse than 20/200 remain at this level or deteriorated further.

Conversion from Ischemic to Non-Ischemic CRVO

Ischemia observed initially may be only relative. Delayed circulation resulting from vascular stasis, as well as inflammation of vessel wall, are reversible with development of collaterals.

Eyes with initially good vision can also become ischemic subsequently and the development of ischemia is more rapid in the first four months.

Neovascularisation

The strongest predictor of iris neovascularisation and angle neovascularisation was extent of capillary nonperfusion seen by FFA. Neovascularisation is first detected by gonioscopy as a fine vascular network adjacent to trabecular meshwork. Next it can be seen in the pupillary margin, causing ectropion uveae. Elevated intraocular pressure associated with NVI/NVA is the hallmark of neovascular glaucoma. Two clock hours of NVI or any NVA is significant anterior segment neovascularisation.

Macular Edema

Macular edema is a major complication of both ischemic and nonischemic CRVO. It is caused by severe capillary leak primarily into the outer plexiform layer. The exact mechanism causing the leakage is unknown, but may be due to vascular congestion, capillary damage or localised inflammatory reactions. Chronic macular edema is associated with poor visual prognosis and needs to be treated.

Macular Ischemia

Inadequate blood supply to macula leads to macular ischemia which causes decreased vision. Presence of ischemia is confirmed ophthalmoscopically by cotton wool spots and attenuated arteries in and around macula.

Fellow Eye Involvement

The existence of systemic risk factors make the fellow eye similarly vulnerable. It has been reported that 5 -10% patients with CRVO develop retinal vein occlusion in the fellow eye.[3-6]

BRVO

SYMPTOMS

The symptoms are painless decreased vision, complete loss of vision or a blind spot in the visual field.

SIGNS

Features are similar to CRVO, except that the changes are localised to single quadrant. Segmentally distributed intraretinal haemorrhages, cotton wool spots, narrowing and sheathing of adjacent artery are seen. At later stages, visual complications may include macular edema, vitreous haemorrhage from neovascularisation, epiretinal membrane or retinal detachment. Clues suggestive of an old BRVO include segmental microvascular abnormalities and intraretinal collateral vessels draining across the median raphe. In nonperfused cases, sclerosis and sheathing of the retinal veins and arteries in the distribution of the occlusion may be observed. Supero-temporal branch retinal vein is most commonly affected.

COMPLICATIONS

The complications after BRVO are macular oedema, neovascularisation, vitreous hemorrhage and retinal detachment.

Macular Edema

It is the most common complication leading to vision loss. It occurs in 5-15% of eyes with BRVO over a period of one year.[7] Interleukin-6 and VEGF have been implicated in the development of macular edema following nonperfused BRVO.

Neovascularisation

Larger areas of persistent retinal nonperfusion can lead to neovascularisation of the retina or disc. The most common site of neovascularisation following BRVO is the retina.

Optic disc neovascularisation is much less common and iris neovascularisation is rare in BRVO. The incidence of retinal neovascularisation is increased in eyes with five disc diameters or more of retinal nonperfusion. Retinal neovascularisation typically develops at the border between perfused and nonperfused retina.

Vitreous Haemorrhage

It may develop after rupture of thin thin, friable neovascular vessels that grow in response to retinal nonperfusion.

Retinal Detatchment

Tractional retinal detachment can form following BRVO, if fibrovascular proliferation develops. Rhegmatogenous retinal detatchments are a rare complication of BRVO. They typically form following posterior retinal breaks caused by fibrovascular proliferation and traction. Nonperfused retina can lead to degeneration and retinal hole formation. Exudative retinal detatchment can occur in area of occlusion and are usually associated with nonperfusion.

Other visually significant complications of BRVO include epiretinal membrane formation, retinal pigment epithelial irregularity, and subretinal scarring.

Fellow Eye Involvement

BVOS reported 9% bilateral involvement.[8, 9]

DIFFERENTIAL DIAGNOSIS

1. **Hypertensive retinopathy**

   Grade 4 hypertensive retinopathy is associated with disc oedema, dilated veins, intraretinal haemorrhages and cotton wool spots. However, it is bilateral and symmetrical. AV crossing changes are prominent. Macular oedema is rare.

2. **Diabetic retinopathy**

   Intraretinal haemorrhages and cottonwool spots are seen, but optic disc oedema is absent, venous pulsations are present and it is bilateral.

3. **Papilloedema**

   Hemorrhages are not as extensive as in CRVO and it is also bilateral.

4. **Ocular ischemic syndrome**

   It is typically associated with mid-peripheral blot-like hemorrhages, iris neovascularization, and ocular pain. OIS is associated with decreased arterial perfusion which is tested by applying light digital pressure on the globe and looking for central retinal artery pulsations.

5. **Hyperviscosity syndromes** (e.g. polycythemia vera, sickle cell disease, leukemia, and multiple myeloma)

6. **Anemic retinopathy**

INVESTIGATIONS

SYSTEMIC INVESTIGATIONS

A systemic workup is not indicated in persons older than 60 years of age with known systemic vascular risk factors for CRVO. Younger patients are more likely to have predisposing conditions resulting in thrombotic disease. A limited systemic work up may be considered in those with a prior occlusion in fellow eye, prior systemic thrombotic disease, family history of thrombosis, or other symptoms suggestive of haematologic or rheumatologic condition.

Medical investigations of underlying systemic risk factors should be done. Blood pressure, random blood sugar, lipid profile, ECG, echocardiography, chest x-ray should be done. Erythrocyte sedimentation rate, anti nuclear antibody, anti phospholipid antibody, fasting plasma homocysteine levels must be done.

FFA

FFA is done in vein occlusions after the haemorrhages resolve.

It helps to differentiate between ischemic and nonischemic type. In ischemic CRVO, the extent of capillary nonperfusion is more than 10 disc diameters. It also helps to evaluate capillary nonperfusion areas at the posterior pole and in the periphery. It can find out new vessels, collaterals, the site of obstruction, macular ischemia and macular edema.

OCT

This investigative modality is used for diagnosis and follow-up. It is a non-invasive technique. It can be used to quantify macular oedema and make comparisons over time. It can also demonstrate the presence of epiretinal membranes.

ERG

In CRVO, ‘b’ wave amplitude is reduced to less than 60% of the normal mean value or as compared with the fellow normal eye. The ‘b’ wave arises from the bipolar cells and Mueller cells in the inner nuclear layer which gets its blood supply from retinal circulation. Hence ‘b’ wave is reduced in CRVO while the ‘a’ wave arising from the photoreceptors which gets blood supply from the choroidal circulation is not affected. As a result, the b/a ratio is reduced in CRVO.

VISUAL FIELDS

Visual fields may be variable in CRVO while BRVO can have quadrantic field defects or scotomas

NEUTRAL DENSITY FILTERS

It is used to quantify RAPD, which may be present in ischemic CRVO.

MANAGEMENT

Medical Therapy

Identification and treatment of systemic vascular risk factors, such as systemic hypertension and diabetes mellitus is important. Prompt initiation of treatment for underlying medical conditions may avert the progression or resolve the existing occlusion.[10]

Anticoagulants, fibrinolytic agents and antiplatelet drugs appear logical , but results from trials have been disappointing, with limited evidence of any benefit, owing to adverse effects of retinal and vitreous hemorrhage.

Oral petoxifylline is a vasodilator and improves perfusion to occluded vessels. It has been tried in combination with systemic hemodilution.

Several studies have suggested a beneficial effect of hemodilution as a therapy in the early phase of RVO. Hemodilution is expected to prevent the slowdown of blood circulation and its complications by dramatically lowering blood viscosity. Hemodilution is recommended in recent-onset CRVO and BRVO when there are no contraindications to treatment.[11]

Systemic steroids and immunosupressives are indicated in inflammatory venous occlusions.

MANAGEMENT OF CRVO

Classification of CRVO Subtypes

The first step in CRVO management is represented by the differential diagnosis between nonischemic CRVO and ischemic CRVO, with an evaluation of nonperfused capillary areas at the posterior pole and of the presence/extension of nonperfused zones at the periphery.

In nonischemic CRVO, major vision-threatening complications are macular edema and conversion to ischemia.

In ischemic CRVO, a major vision-threatening complication is the development of ocular neovascularization, especially in the anterior segment of the eye (iris and angle, up to neovascular glaucoma).

Fluorescein angiography showing extensive (more than 10 disc areas of nonperfusion) retinal capillary nonperfusion suggests ischemic CRVO.
The presence of macular ischemia is seen as enlargement of the foveal avascular zone on fluorescein angiography.

Management of nonischemic CRVO

In patients with nonischemic (well-perfused) CRVO and good VA (better than 20/40), the prognosis is favourable and monitoring is possible. In this case, no immediate therapy needs to be advised, but there still is a need to screen all new patients for vascular risk factors such as hypertension, dyslipidemia and diabetes. Treatment of these underlying causes is of paramount importance to prevent complications. An investigation of other risk factors should be considered only when the clinical history suggests their presence, in the absence of other obvious etiology. Local factors predisposing to or associated with CRVO, such as open-angle glaucoma, should be ruled out and treated appropriately to reduce the risk of progression to a more ischemic state.

Monitoring during follow-up is aimed at identifying persistent macular edema and/or conversion to ischemic CRVO. Key elements in the clinical examination include VA assessment, biomicroscopy and OCT. Fluorescein angiography should be performed whenever there is doubt regarding progression, or to assess the degree of ischemia.

Patients should be monitored monthly for the first 3 months, and then every 2 months for the first year. During this monitoring period, patients should be instructed to return promptly whenever they notice a decrease in vision, a possible indication of macular edema, or the conversion to ischemic CRVO.

Nonischemic CRVO and VA of 20/40 or Less

In nonischemic CRVO, with a VA which is 20/40 or less, one should search for macular edema. In case of macular edema, treatment should be initiated rather than observation.

Management of Macular Edema in Well-Perfused CRVO

1. Laser Photocoagulation

In the CVOS, even though grid laser photocoagulation was able to reduce macular edema, there was no statistically significant VA benefit, except for the younger patient group. Thus, grid laser photocoagulation is not currently indicated.[12]

Current treatment options include corticosteroids and antivascular endothelial growth factor (VEGF) approaches. Eyes affected by macular edema secondary to CRVO must be considered for treatment whenever VA is lower than 20/40.

2. Corticosteroids Approach

The rationale for the use of steroids to treat macular edema is related to their ability to reduce capillary permeability, and to inhibit the expression of VEGF gene and the metabolic pathway of VEGF. The corticosteroids used are Dexamethasone and Triamcinolone acetonide.

2.1. Dexamethasone

Dexamethasone has been used for a long time as a potent corticosteroid that decreases inflammatory mediators implicated in macular edema. Dexamethasone is highly soluble and has a short half-life following intravitreal injection. To provide a sustained delivery of dexamethasone, a slow release, biodegradable implant (Ozurdex; Allergan) was developed, providing medication for up to 6 months at the posterior pole following implantation in the vitreous cavity. Its therapeutic effects on macular edema associated with RVO were investigated in a 6-month, randomized, controlled clinical trial (the Ozurdex GENEVA study).[13] A prefilled, single-use applicator containing 0.7 mg of dexamethasone in a slow-release polyglycolate-acetate implant allows the insertion of the drug. The Ozurdex GENEVA study demonstrated that the biodegradable implant containing 0.7 mg of dexamethasone (Ozurdex) resulted in improved VA, revealing a peak effect after 2 months and a progressive decline to baseline values at 6 months. The data on safety showed a low cataract rate and low rates of intraocular pressure increases. The study was also able to show that early treatment of macular edema was more beneficial than delayed treatment in restoring VA. It is likely that a dexamethasone implant should be reinjected more frequently, following the individual response of each patient over the follow-up. Ozurdex has received FDA and EU approval for the treatment of adult patients with macular edema following CRVO.

2.2 Triamcinolone acetonide

Triamcinolone acetonide has a number of side effects including the development of cataract and raised intraocular pressure. The presence of benzyl alcohol also leads to an increased risk of sterile endophthalmitis. The multicenter SCORE trial[14-16] has confirmed the beneficial effects of intravitreal triamcinolone acetonide for the treatment of macular edema associated with nonischemic CRVO. For the purpose of this study, a preservative-free triamcinolone preparation (Trivaris; Allergan) was used. The odds of achieving the primary outcome were 5.0 times greater both in the 1-mg group and in the 4-mg group than in the observation group ($p = 0.001$). There was no difference identified between the 1-mg and 4-mg groups ($p = 0.97$). The 1-mg dose showed a better safety profile compared with the 4-mg dose, with lower incidence rates of raised intraocular pressure, cataract formation, progression and surgery

At present there is no evidence to suggest that the visual and anatomical responses seen with Trivaris in the SCORE study would be achieved with off-label intravitreous triamcinolone acetonide preparations such as Kenalog.

3. Anti-VEGF Approach

Anti-VEGF agents reduce the capillary permeability and hence are used in the treatment of macular oedema due to CRVO. Intravitreal anti-VEGF administration of ranibizumab, bevacizumab and pegaptanib have been investigated.

3.1. Ranibizumab

Ranibizumab is a pan-VEGF blocker (Lucentis ; Novartis) proved to be effective in the CRUISE trial.[17] Overall, the 12-month results suggested that the visual gain could be sustained. Moreover, earlier treatment could lead to a greater functional improvement than delayed therapy. Ranibizumab 0.5 mg has received a license for the treatment of macular edema following RVO.

3.2. Bevacizumab

Bevacizumab is a pan-VEGF blocker (Avastin ; Roche) and is unlicensed for intraocular use. Although there is no randomized clinical trial involving bevacizumab in RVO, many uncontrolled case series have reported that intravitreal administration can lead to a VA improvement and resolution of macular edema. However, because of the variation in dosing and treatment regimens among these studies, both long-term outcomes and safety data remain unclear. The drug has the advantage to be less costly, which has helped its widespread use.

3.3. Pegaptanib

Pegaptanib is a selective anti-VEGF 165 blocker (Macugen; Pfizer). The first multicenter randomized study on the effect of anti-VEGF therapy in the treatment of RVO was designed to evaluate the efficacy of pegaptanib sodium. A phase II trial[18] indicated that 0.3 mg intravitreal pegaptanib given every 6 weeks over a 6-month follow-up improved VA by approximately 7 letters at 6 months. The best treatment regimen and response to treatment in the long run still remain unclear.

Recommendations for Further Follow-Up

Follow-up after the initial 6 months will depend upon whether steroid or anti-VEGF treatment was initiated for macular edema, but it will normally be required for up to 2 years in uncomplicated cases. The eyes should be monitored for conversion to ischemia and for occurrence/recurrence of macular edema. The development of disk collaterals and the resolution of macular edema should lead to discharge from close clinical supervision, but the risk of conversion remains present and should be explained to the patients.

Recurrence or persistence of macular edema, should lead to a decision of reinjection during the follow-up period. Additional laser photocoagulation could be suggested for nonresponding or partially responding patients, or for patients not complying with multiple reinjections.

Management of Ischemic CRVO

In patients with ischemic CRVO, primary evaluation should assess the presence of macular perfusion as well as the existence of neovascularization.

1. Macular Perfusion

In cases with macular edema and a still perfused macula, the same treatment as outlined above for cases of nonischemic CRVO should be initiated. In case of a macula which is nonperfused, treatment should be initiated even if the expectation of visual improvement remains limited.

2. Peripheral Nonperfusion

Ischemic CRVO is usually characterized by peripheral retinal nonperfusion greater than 10 disc diameters, as evaluated by FFA. Development of ocular neovascularization is directly related to the extent of nonperfusion. Cases with extensive retinal nonperfusion, or with limited compliance, may be considered for early PRP in an attempt to block the development of ocular neovascularization. In less severe cases, scatter treatment directed at the nonperfused areas may be sufficient. In addition to treatment for macular edema, patients with ischemic CRVO without neovascularisation should be followed up at least monthly, undergoing VA, biomicroscopy, OCT and FFA (if needed). The corneal angle and iris should also be examined in an attempt to discover early neovascular tufts.[19]

Management of Neovascularization

1. Anterior Segment Neovascularization

Whenever anterior segment neovascularization (angle and/or iris neovascularization) is identified, evidence based medicine supports the administration of PRP. In particular, the extent of anterior segment neovascularization (which explicitly requires PRP) has been defined as any angle neovascularization and/or 2 clock hours of iris neovascularisation.

A complete PRP can be achieved in single or multiple sessions to cover the entire retina from the periphery to the vascular arcades. PRP generally requires a minimum of 1,500–2,000 burns, or even more, of 500 micron size, with 0.1 second applications. Laser burns should be placed 1 burn width apart with sufficient energy to produce a pale white burn in the retina. Treatment is usually placed trying to avoid areas of retinal hemorrhage. The laser application must begin with the inferior quadrants, may be completed in a few weeks, and can be repeated whenever anterior segment neovascularization fails to regress.

Even though there is no randomized clinical trial of the combined therapy, it is reasonable to administer an intravitreal anti-VEGF agent in association with PRP as it may lead to faster regression of anterior segment neovascularisation.

2. Posterior Segment Neovascularization

Posterior segment neovascularization (retinal and/or optic disc neovascularization) can develop alone or in association with anterior segment neovascularization. It can be treated with PRP as described above. Also, combined treatment with anti-VEGF and PRP may prove useful to effectively control the growth of neovascularization. Monotherapy with intravitreal injection of anti-VEGF agents such as ranibizumab and bevacizumab can lead to a transient regression of ocular neovascularisation.

Patients presenting with widespread ocular neovascularisation should be treated with PRP as soon as possible. Especially in eyes with vitreous hemorrhage, the combined therapy with anti-VEGF agents may prove useful to stop the growing of ocular neovascularization, allowing at the same time the application of prompt PRP in a few sessions (if the vitreous hemorrhage is not too dense, which would then require vitrectomy and endolaser therapy).

3. Neovascular Glaucoma

In the case of established neovascular glaucoma, intravitreal bevacizumab has been shown to cause regression of iris new vessels and decrease angle obstruction. Iris new vessels regress faster after intravitreal bevacizumab with PRP than with PRP alone. Bevacizumab may reduce the need for surgical interventions and serve as a useful adjunct to filtering surgery.

4. Juvenile CRVO

Juvenile CRVO, occurring in people younger than 50 years, seems to be a different entity regarding pathogenesis and clinical course, and should be differentiated from CRVO developing after 50 years of age. In some cases, when the disease is associated with severe systemic disease, patients need to be addressed for complete systemic evaluation, and the prognosis can be guarded.

Juvenile CRVO is often of a non ischemic type, with no clearly identifiable risk factors, and sometimes related to an inflammatory pathogenesis, as shown by the detection of vitreous cells. Visual prognosis is generally better in comparison with ordinary CRVO, even though possible complications include ocular neovascularization and the development of macular edema.

Some evidence exists that systemic steroids can lead to a faster resolution of the disease. Even though there is no randomized clinical trial, it is plausible that intraocular steroids, especially the intravitreal slow-release dexamethasone implant, may prove useful in the management of macular edema secondary to juvenile CRVO.

MANAGEMENT OF BRVO

The management of BRVO has many similarities to the management of CRVO with regard to systemic risk factors, but it presents some important differences because this type of RVO carries a lower risk of progressive worsening and conversion to ischemia and a lower risk of neovascularization.

Thus, the management of BRVO should embrace several targets including: the identification and management of systemic risk factors; a precise classification of the area of inclusion to determine major branch versus tributary branch occlusion; an assessment of the degree of peripheral perfusion and degree of macular ischemia, and the institution of treatment according to sight-threatening complications (mainly persistent macular edema and neovascularization).

BRVO with Perfused Periphery and Normal VA

In case of a BRVO with a perfused periphery and normal VA, the prognosis is favorable and monitoring is possible. In this case, no therapy needs to be advised. In the follow-up examination, one should look for significant macular edema by VA examination, biomicroscopy and OCT. If in doubt, fluorescein angiography should be performed. Patients should be monitored monthly for the first 3 months, and then every 2 months for the first year. During this monitoring period, patients should be instructed to quickly report whenever they notice a loss of VA, which may indicate the development of macular edema.

BRVO with Perfused Periphery and Symptomatic VA Decline

In cases of BRVO and symptomatic decline in VA, an assessment should be performed for the existence of macular edema. If there is macular edema seen by OCT, treatment should be considered according to the following outline. Special attention should be paid to macular BRVO, a subtype of BRVO involving a small vein draining a sector of the macular region, which has a more favorable natural course, not requiring treatment in most cases.

1.Laser photocoagulation

For many years, grid laser photocoagulation has been the standard care and recommended for patients with macular edema associated with branch vein occlusion who met the BVOS eligibility criteria (VA of 20/40 or less, persistent macular edema lasting for 4 months or longer, and resorption of macular hemorrhages).[8, 9] Recently, based on the results of a new prospective, double-masked randomized trial, the SCORE study[20], it was recommended that grid photocoagulation should remain the treatment of choice for eyes with vision loss associated with macular edema secondary to BRVO. The study concluded that no difference was found in VA at 12 months in the standard care group (grid laser photocoagulation) compared with the triamcinolone groups. Moreover, rates of adverse events (particularly elevated intraocular pressure and cataract) were highest in the 4-mg triamcinolone group.

2. Intravitreal Drugs

2.1. Intravitreal Steroids

2.1.1. Dexamethasone

The Ozurdex GENEVA study showed that the maximum effect was at day 60 and a decrease in effect began at day 90, but was still persistent at day 180. The second injection was even slightly more effective than the first injection. No adverse events were related to the injection, with a very low cataract rate and very low rates of persistent intraocular pressure increase.

2.1.2. Intravitreal triamcinolone acetonide

Recently, the SCORE trial[20] compared the efficacy and safety of 1-mg and 4-mg doses of preservative-free intravitreal triamcinolone with standard care (grid photocoagulation) for eyes with vision loss associated with macular edema secondary to BRVO, and concluded that there was no difference identified in VA at 12 months for the standard care group compared with the triamcinolone groups; however, rates of adverse events (particularly elevated intraocular pressure and cataract) were highest in the 4-mg group.

2.2. Intravitreal Anti-VEGF Drugs

Intravitreal anti-VEGF administration has been tried with ranibizumab, bevacizumab and pegaptanib. Ranibizumab was proved to be effective for macular oedema secondary to BRVO in the BRAVO trial.

Management of BRVO with Peripheral Nonperfusion

In these cases, the perfusion of the macula should be assessed. If it is perfused, treatment should be contemplated as outlined above. Laser treatment of the peripheral areas of nonperfusion can be considered if the nonperfused area is very extensive.

If the macula is not perfused, again, treatment should be contemplated as outlined above, with informed consent of patients as the prognosis can be bleak.

In cases of BRVO with peripheral neovascularization, intravitreal therapy should be initiated, followed by scatter laser aimed at the area of the occluded vein.

PREVENTION

Only few studies have addressed the prevention of recurrence of RVO in the same eye, or of the development of RVO in the contralateral eye. So far, none of these studies have shown any benefit. Available data support the concept that recurrence of RVO may be reduced by medical treatment of underlying cardiovascular risk factors.

Historically, hormone replacement therapy was contraindicated and discontinued following central vein occlusion. Currently, the decision about whether to continue hormone replacement therapy in women with RVO should be made on a case-by-case basis.

SURGICAL APPROACHES TO TREATING RVO

The focus of surgical treatment is either the occluded retinal vein itself or the macular edema. Many surgical treatment modalities have been reported for RVO. Of the following common surgical approaches, only a few are frequently utilized:

(1) Radial optic neurotomy (RON);

(2) Chorioretinal venous anastomosis;

(3) Vitrectomy with or without internal limiting membrane (ILM) peeling;

(4) Injection of tissue plasminogen activator (t-PA) into the lumen of a retinal vein via retinal vein cannulation;

(5) arteriovenous sheathotomy

1. Radial Neurotomy

Combined pars plana vitrectomy with transvitreal incision of nasal sclera ring, radial to the optic nerve and parallel to nerve fibre layer, releases the pressure on central retinal vein and the scleral outlet. This procedure addresses the compartmental syndrome. Radial optic neurotomy also induces development of optociliary venous anastomosis or retinochoroidal shunts, leading to increased retinal venous outflow.

Reported complications of radial optic neurotomy include optic nerve damage, temporal visual feild defect, injury to central retinal artery or vein, subretinal hemorrhage, vitreous hemorrhage, globe perforation, peripapillary retinal detachment, choroidal neovascularisation and anterior segment neovascularization.[21-23] The benefits of RON appear to be controversial and its efficacy remains to be proven in prospective randomized clinical studies.

2. Chorioretinal Venous Anastomosis

In this method, the occluded central retinal vein is bypassed by creating a chorioretinal anastamosis between nasal branch of central retinal vein with the choroidal circulation. It prevents the development of retinal ischemia. Visual acuity may improve as a result of resolution of macular oedema and maintenance of retinal perfusion. This is done surgically or by using laser.[24] Laser energy delivered through Nd:YAG or argon laser is directed at branch retinal vein to rupture the posterior vein wall and Bruch's membrane.

Complications include intraretinal, subretinal and vitreous hemorrhage, secondary neovascularisation, fibrovascular proliferation and tractional detachment

3. Vitrectomy with or without ILM Peeling

Pars plana vitectomy can be done in cases of nonclearing vitreous hemorrhage. It can be combined with epiretinal membrane peeling with endolaser photocoagulation if needed. Relieving the vitreous traction over the macula by means of vitrectomy induced PVD improves macular oedema.

A statistically significant improvement in patients after vitrectomy with gas/air tamponade for macular edema caused by BRVO was reported .[25-26]

4. Arteriovenous Sheathotomy

It is an attempt to decompress the involved venule by separating the overlying retinal artery from the underlying branch vein by sectioning the shared adventitial sheath. Patients who have macular oedema recalcitrant to grid may be candidates for AV sheathotomy. More recently, a bimanual technique of arteriovenous sheathotomy followed by intravitreal recombinant t-PA is done.[27]

Complications include nerve fibre layer defects, vitreous hemorrhage, retinal tear and retinal detachment.

AIM

- To study the commonest etiology for retinal vein occlusion in patients attending Ophthalmology outpatient department.
- To study the response to treatment in terms of visual acuity, macular thickness and intraocular tension.
- To study the FFA and OCT features of retinal vein occlusions.
- To study the prevalence of retinal vein occlusion in relation to etiology, age, gender, laterality, quadrant of involvement and risk factors
- To assess the risk factors associated with retinal vein occlusion and the factors associated with better visual outcome

METHODOLOGY

All new and review cases attending Ophthalmology outpatient department diagnosed to have retinal vein occlusion were included in the study. A total of 51 eyes of 51 patients were included in this study.

PROCEDURE

A detailed history, vision, intraocular tension, slit lamp examination and fundus examination were done. Fundus fluorescein angiography (FFA) and Optical coherence tomography (OCT) were done in appropriate cases. Random blood sugar, BP, lipid profile and cardiac evaluation were done. The treatment was tailored according to individual patient. The underlying systemic disease was treated. For macular edema, either macular grid laser or intravitreal anti-VEGF injection was given. If there were new vessels or significant capillary nonperfusion areas seen on FFA, laser photocoagulation was done.

INCLUSION CRITERIA

All patients diagnosed to have retinal venous occlusion were included in the study.

EXCLUSION CRITERIA

Patients diagnosed to have vein occlusion due to vasculitis were excluded from the study.

TREATMENT

Treatment was tailored according to individual patient.

The underlying systemic disease was treated. If visual acuity was more than 6/12, with no macular pathology and no significant capillary non perfusion areas, careful observation was done. If vision was less than 6/12, search for macular edema or ischemia was done. For macular edema, either macular grid laser or intravitreal anti-VEGF injection was given. If there were new vessels or significant capillary nonperfusion areas seen on FFA (more than 5 disc diameter areas for BRVO and more than 10 disc diameter areas for CRVO), laser photocoagulation was done with or without intravitreal anti-VEGF. The anti-VEGF injection was repeated if required.

FOLLOW UP

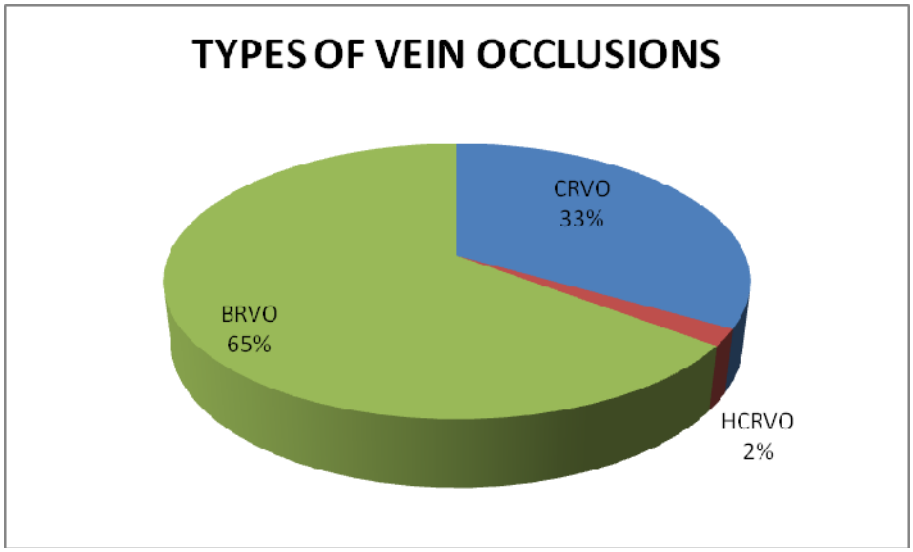
Follow up was done at 4, 12 and 24 weeks.

RESULTS

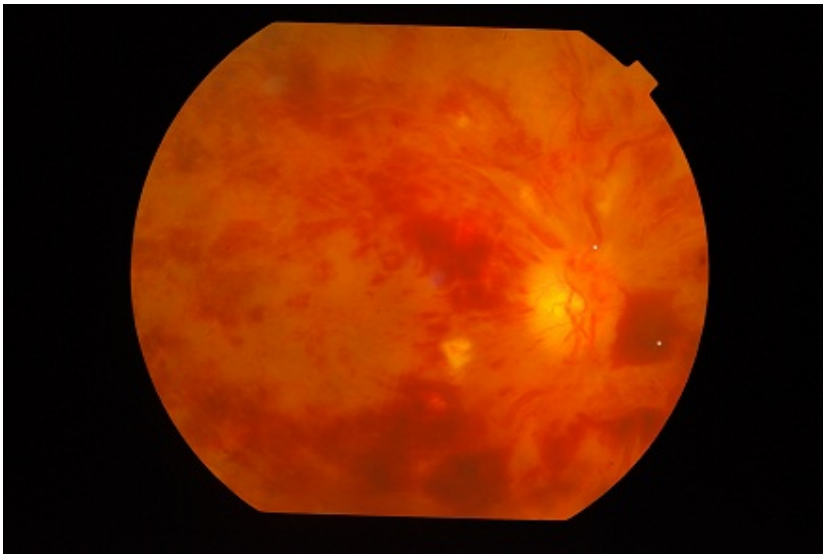
We studied retinal vein occlusions in 51 eyes of 51 patients who presented to Ophthalmology outpatient department. Of these, 17 had CRVO, 1 had HCRVO and 33 had BRVO.

TABLE 1 PERCENTAGE OF VARIOUS TYPES OF RVO

Types of Vein Occlusions	**Number (%)**
CRVO	17 (33%)
HCRVO	1 (2%)
BRVO	33 (65%)

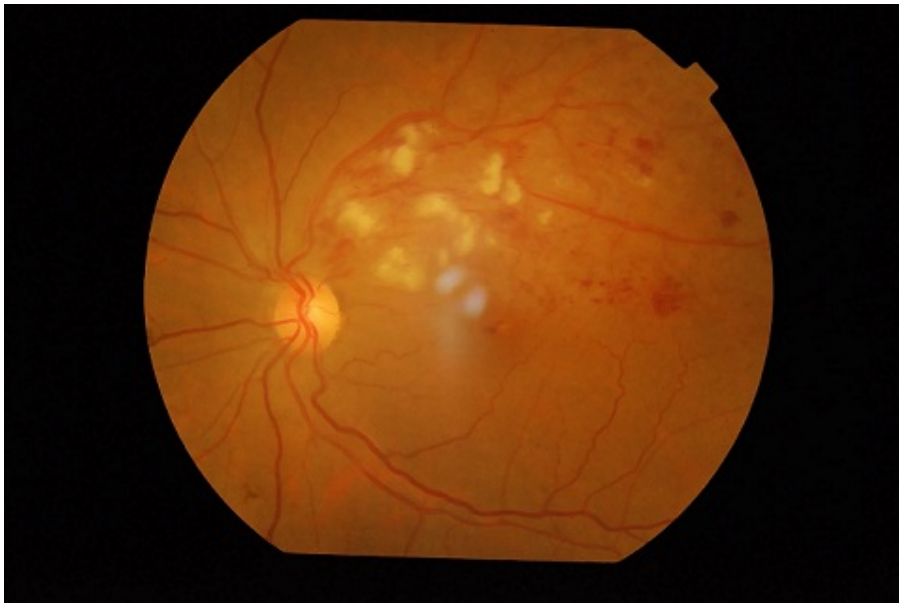


CRVO



HCRVO

ST BRVO



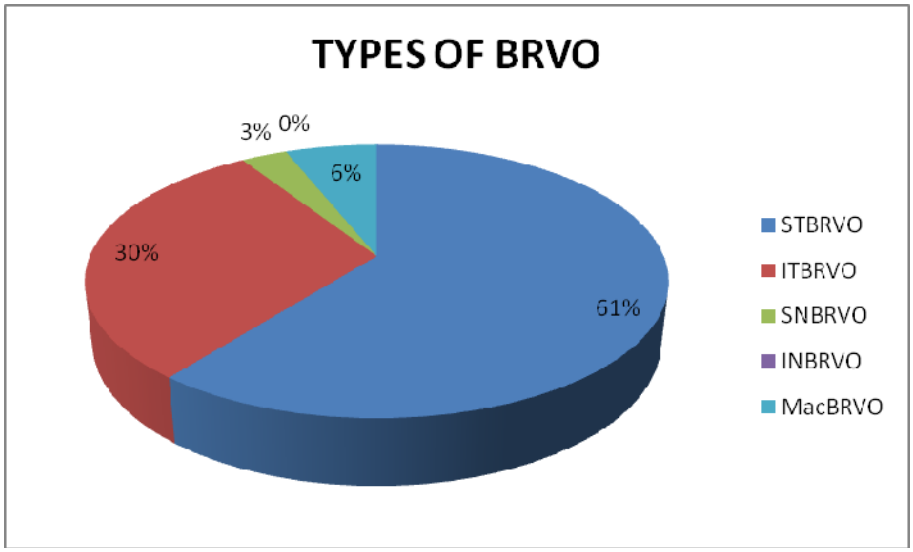
MACULAR BRVO

TYPES OF BRVO

Of the 33 patients with BRVO studied, 20 had ST BRVO, 10 had IT BRVO, 1 had SN BRVO and 2 had macular BRVO.

TABLE 2 TYPES OF BRVO

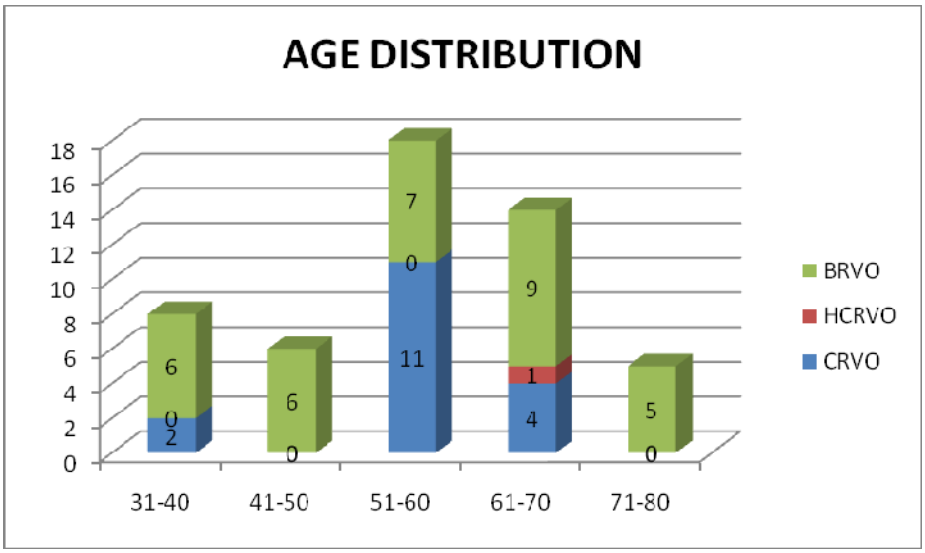
Type of BRVO	Number (%)
ST BRVO	20 (60.6%)
IT BRVO	10 (30.3%)
SN BRVO	1 (3.03%)
IN BRVO	0 (0%)
Mac BRVO	2 (6.06%)



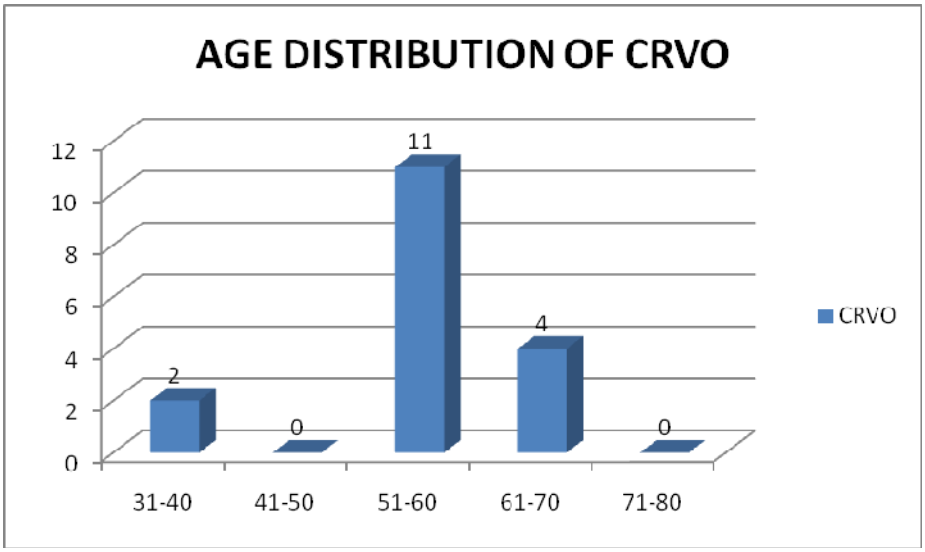
AGE DISTRIBUTION

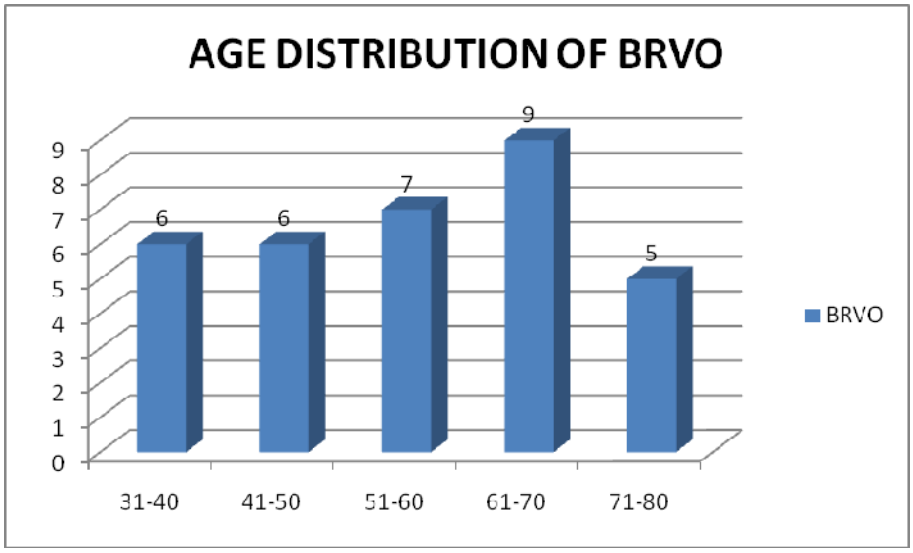
TABLE 3 AGE DISTRIBUTION

Age in years	CRVO (17)	HCRVO (1)	BRVO (33)	Total (51)
31-40	2	0	6	8 (15.6%)
41-50	0	0	6	6 (11.7%)
51-60	11	0	7	18 (35.2%)
61-70	4	1	9	14 (27.4%)
71-80	0	0	5	5 (9.8%)



The mean age of the patients included in the study was 56.27 ± 11.28 years, ranging from 35 to 76 years. Most of the patients, 32(64.7%) were in the age group of 51-70 years. Patients of age less than 50 years were just 14 (27.5%), while patients more than 50 years were 37 (72.5%). Retinal vein occlusion is therefore more common in the elderly.





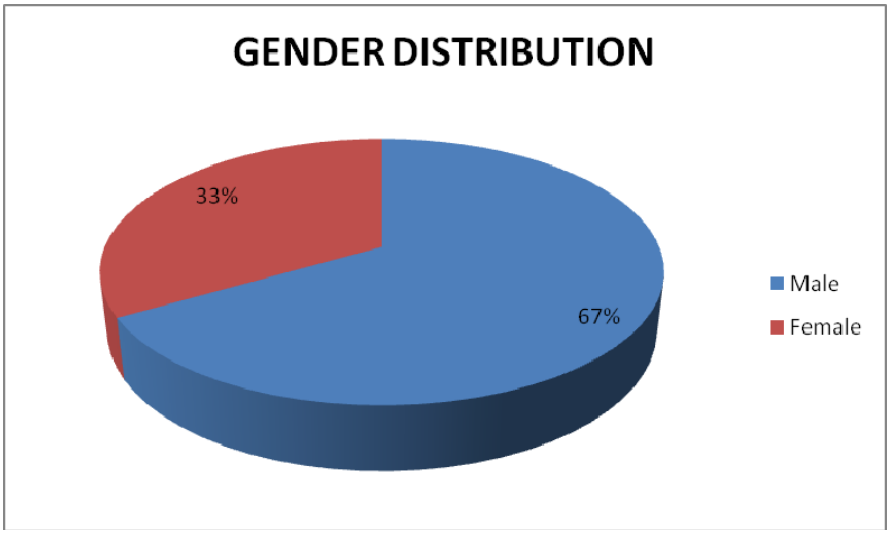
A subgroup analysis revealed that most of the patients with CRVO were in the 6^{th} (11, 64.7%) and 7^{th} (4, 23.7%) decades, while patients were more evenly distributed in the BRVO group.

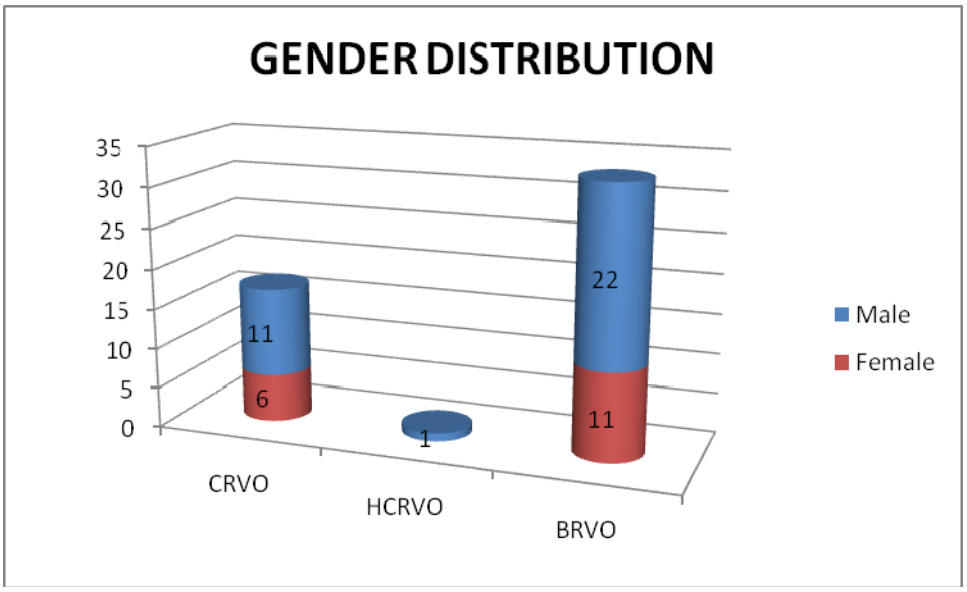
GENDER DISTRIBUTION

In our study, we found male preponderance for both CRVO and BRVO, of 64.7% and 66.7% respectively. The male preponderance could be explained by the increased incidence of smoking among males.

TABLE 4 GENDER DISTRIBUTION

Gender	**CRVO**	**HCRVO**	**BRVO**	**TOTAL**
Male	11	1	22	34
Female	6	0	11	13


GENDER DISTRIBUTION
33%
67%
Male
Female


GENDER DISTRIBUTION
35
30
25
20
15
10
5
0
11
6
1
22
11
CRVO
HCRVO
BRVO
Male
Female

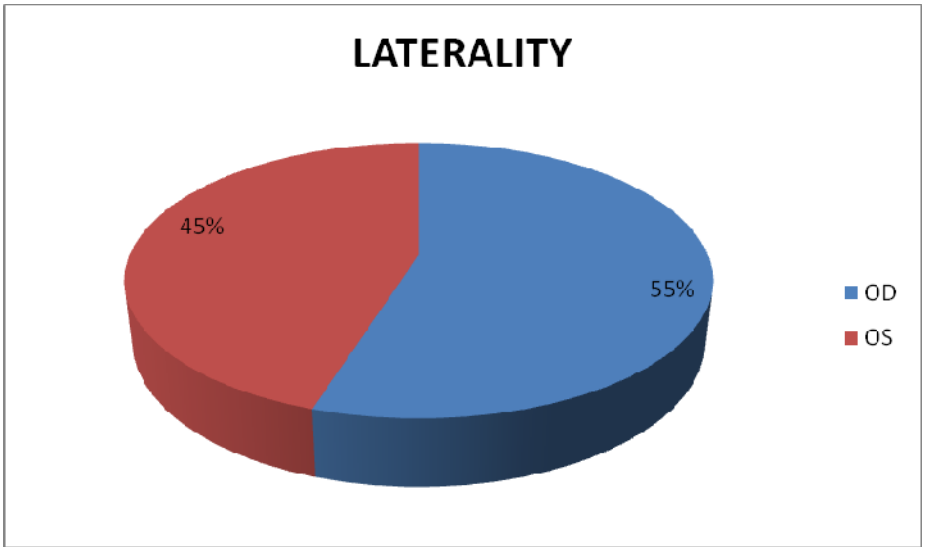
LATERALITY

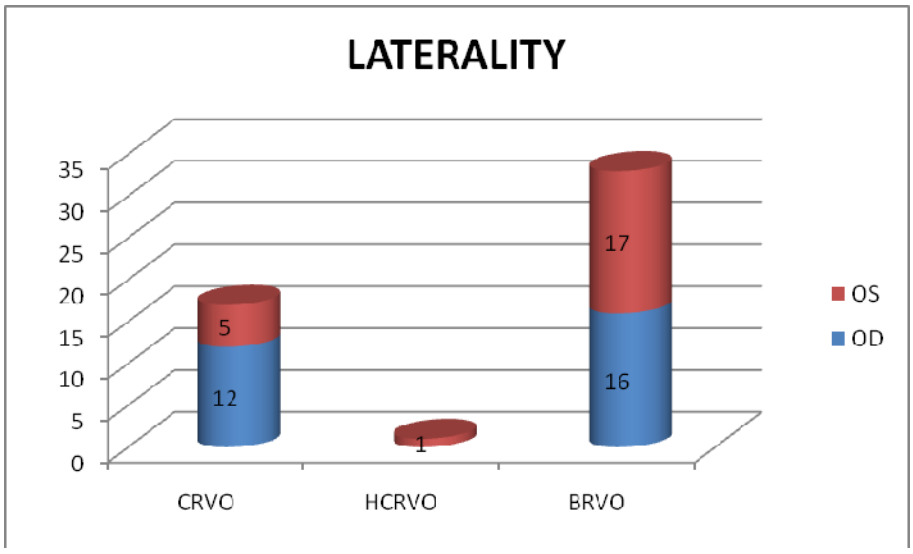
All the eyes studied had unilateral involvement. Twenty eight (54.9%) were right eyes and twenty three (45.1%) were left eyes.

We found that 12 (70.6%) of CRVO involved the right eye. In case of BRVO, there was no difference in involvement of either eye.

TABLE 5 LATERALITY

Laterality	**OD**	**OS**
CRVO	12	5
HCRVO	0	1
BRVO	16	17
Total	28	23


LATERALITY
45%
55%
OD
OS

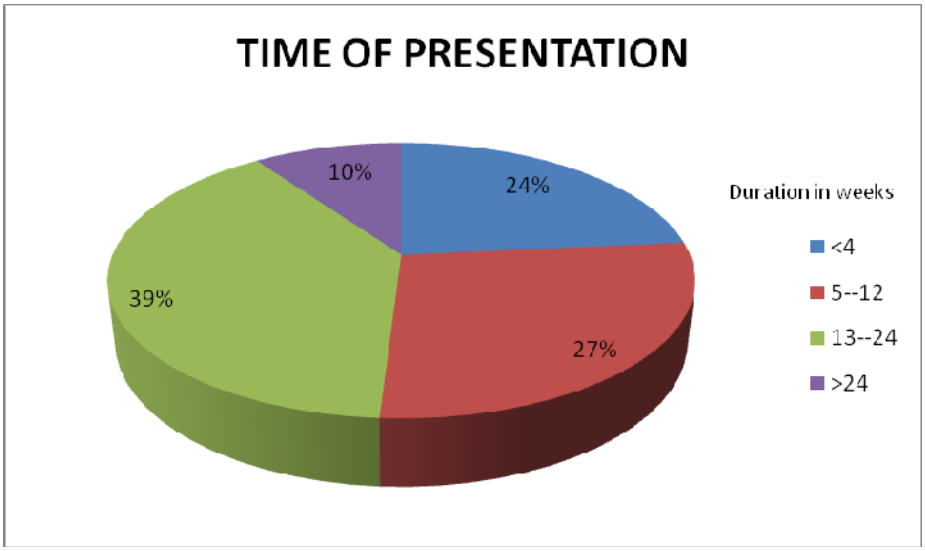

LATERALITY
35
30
25
20
15
10
5
0
5
12
1
17
16
CRVO
HCRVO
BRVO
OS
OD

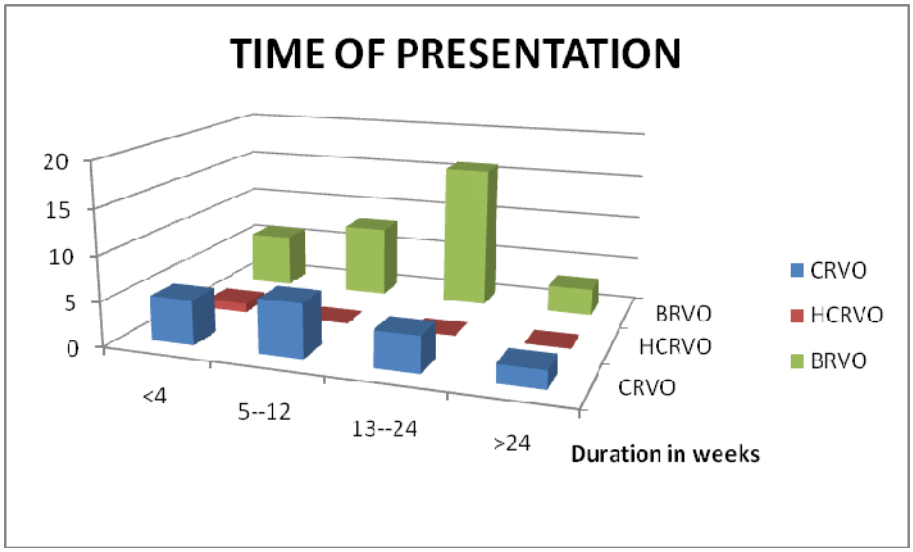
TIME OF PRESENTATION

Of the CRVO patients, 5 (29.4%) presented within 4 weeks of the onset of venous occlusion, while 6 (18.1%) BRVO patients presented within 4 weeks. The rest of the patients presented after more than 1 month of the symptoms. This may be because of the fact that ours is a tertiary care ophthalmic centre, referred cases from peripheral hospitals came to our hospital a prolonged period after the onset of disease. More BRVO cases presented later than a month which may be because BRVO did not significantly affect vision when compared to CRVO.

TABLE 6 DURATION OF DIMINISHED VISION

Type	**<4 WEEKS**	**5-12 WEEKS**	**13-24 WEEK**	**>24 WEEKS**
CRVO	5 (29.4%)	6 (35.2%)	4 (23.5%)	2 (11.7%)
HCRVO	1	0	0	0
BRVO	6 (18.1%)	8 (24.2%)	16 (48.4%)	3 (9%)
Total	12 (23.5%)	14 (27.4%)	20 (39.2%)	5 (9.8%)

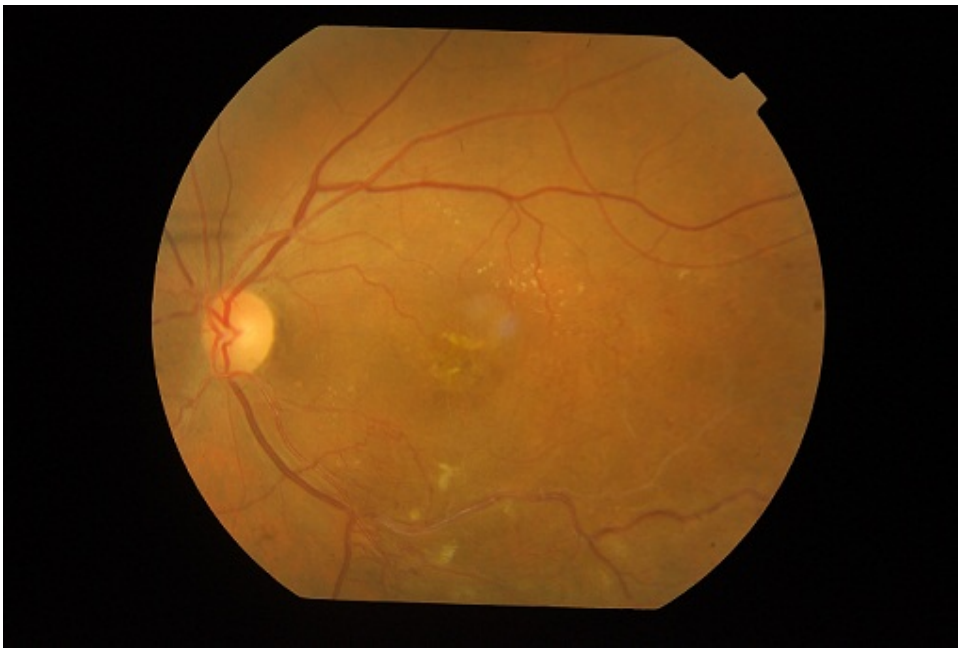




Three of our CRVO patients presented after 20 weeks and had neovascular glaucoma. One of them had high IOP of 48 mm Hg which was managed medically. Susequently he underwent anti-VEGF injection and panretinal photocoagulation. Another patient also received anti-VEGF injection and panretinal photocoagulation, while the third person underwent panretinal photocoagulation alone.

FRESH IT BRVO

OLD IT BRVO



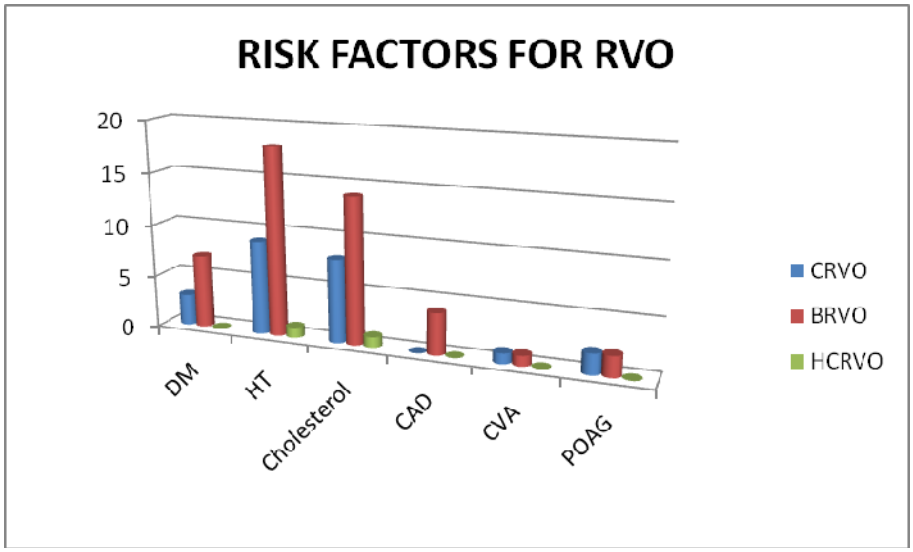
RISK FACTORS FOR VEIN OCCLUSION

The commonest comorbid condition was hypertension, which was found in 28 (53%) patients. Diabetes mellitus was present in 10 (19.6%) patients. Twenty three (45%) patients had serum cholesterol more than 200 mg%. Four patients (7.8%) had a coronary artery disease (CAD) while 2 patients (3.9%) had a history of cerebro-vascular accident (CVA), Four patients (7.8%) had primary open angle glaucoma.

TABLE 7 RISK FACTORS

Risk Factor	**CRVO**	**HCRVO**	**BRVO**	**Total**
DM	3 (17.6%)	0	7 (21.2%)	10 (19.6%)
HT	9 (52.9%)	1	18 (54.5%)	28 (54.9%)
Hypercholestolemia	8(47%)	1	14 (42.4%)	23 (45%)
CAD	0	0	4 (12.1%)	4 (7.8%)
CVA	1 (5.8%)	0	1 (3%)	2 (3.9%)
POAG	2 (11.7%)	0	2 (6%)	4 (7.8%)

Among CRVO cases, 3 (17.6%) had diabetes mellitus, 9 (52.9%) were hypertensive, 8 (47%) had hypercholesterolemia, 1 (5.8%) had a history of CVA and 2 (11.7%) had POAG. Among the BRVO patients, 7 (21.2%) were diabetic, 18 (54.9%) were hypertensive. 14 (42.4%) had hypercholestolemia, 4 (12.1%) had CAD, 1(3%) had CVA and 2 (6%) had POAG



The mean random blood sugar was 133 ± 36 mg%. The mean systolic blood pressure was 144 ± 21 mmHg and the mean diastolic blood pressure was 90 ± 10 mmHg. The mean serum cholesterol was 195 ± 33 mg%.

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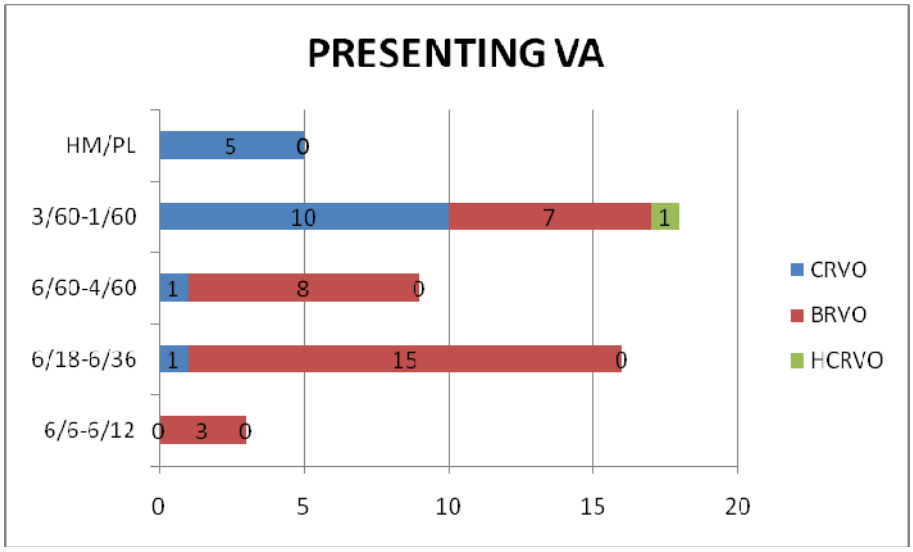
PRESENTING VISUAL ACUITY

Among CRVO patients, 15 (88.2%) had vision less than 3/60 at presentation, while only 7 (21.2%) BRVO patients had such poor vision. Also, 18 (54.5%) BRVO patients had presenting vision of 6/36 or better which was present in only 1 (5.8%) patient with CRVO.

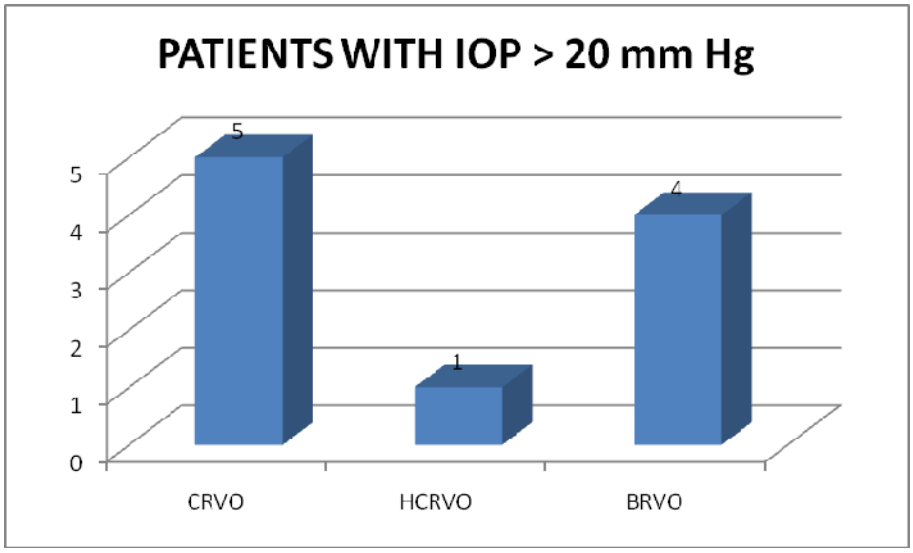
TABLE 8 PRESENTING VISUAL ACUITY

VA	CRVO	HCRVO	BRVO	TOTAL
6/6-6/12	0	0	3 (9%)	3 (5.8%)
6/18-6/36	1 (5.8%)	0	15 (45.4%)	16 (31.3%)
6/60-4/60	1 (5.8%)	0	8 (24.2%)	9 (17.6%)
3/60-1/60	10 (58.8%)	1	7 (21.2%)	18 (35.2%)
HM/PL	5 (29.4%)	0	0	5 (9.8%)



INTRAOCULAR PRESSURE

The mean intraocular pressure at presentation was 16.8 ± 5.4 mm Hg. Ten patients (19.6%) had intraocular pressure more than 20 mm Hg and were managed medically. Four patients had primary open angle glaucoma and 3 patients had neovascular glaucoma secondary to the vascular occlusion. CRVO patients had higher IOP (18.9 mm Hg) than BRVO patients (15.6 mm Hg).



FFA FINDINGS

Fundus fluorescein angiography was done in 50 patients. One patient with vitreous hemorrhage did not undergo FFA.

Eight (50%) CRVO patients had capillary non perfusion areas more than 10 disc diameter area, 6 (37.5%) had new vessels, and 2 (12.5%) had macular oedema. Macular ischemia was noted in 4 (25%) of patients with CRVO.

Of the BRVO patients, 8 (24.2%) had capillary non perfusion more than 5 disc diameter area, 2 (6%) had collaterals, 8 (24.2%) had new vessels, 10 (30.3%) had macular oedema and 2 (6%) had macular iscemia.

The patient with HCRVO had macular ischemia and collateral formation.

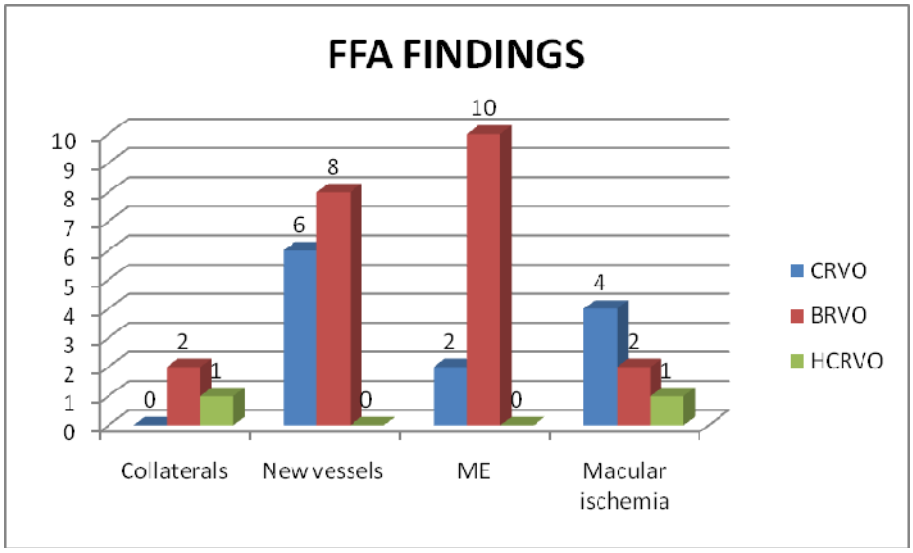
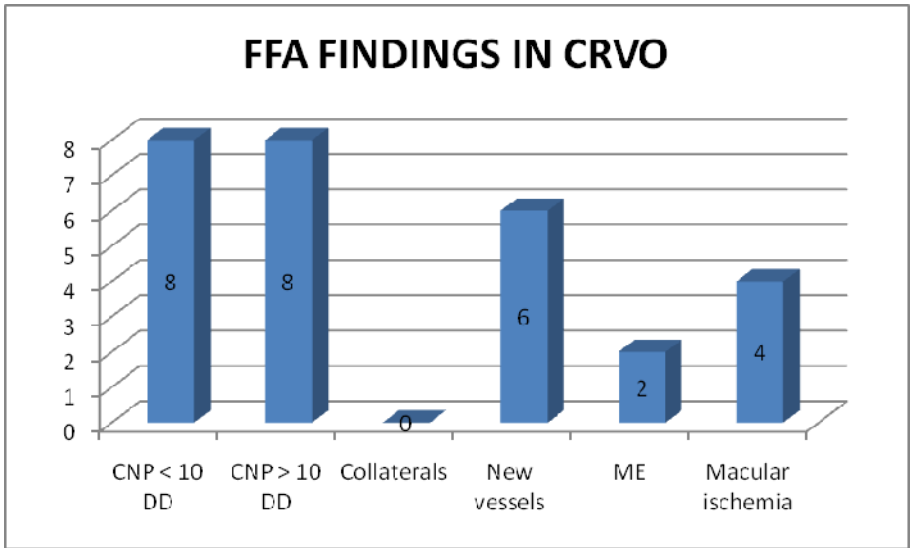
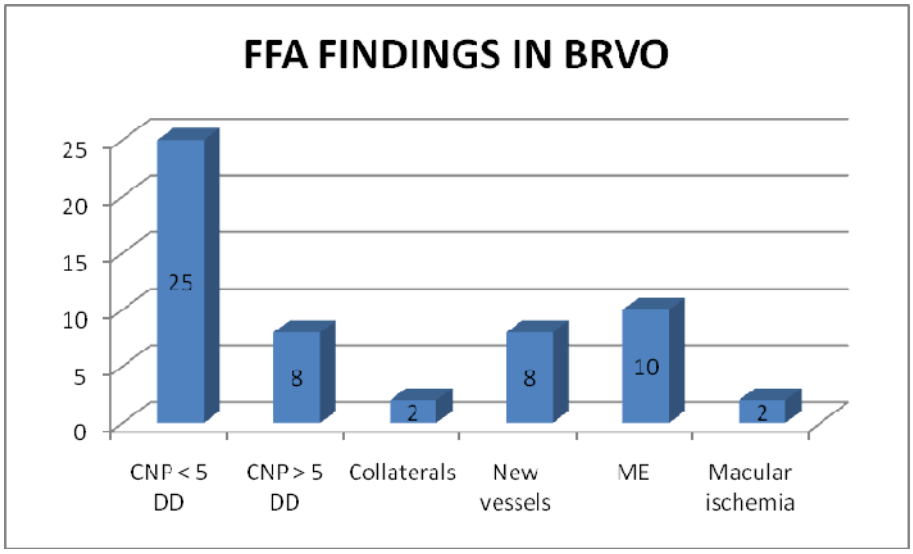


TABLE 9 FFA FINDINGS

FFA Finding	CRVO (16)	HCRVO (1)	BRVO (33)	TOTAL (50)
SIGNIFICANT CNP	8 (50%)	0	8 (24.2%)	16 (32%)
COLLATERALS	0	1	2 (6%)	3 (6%)
NEW VESSELS	6 (37.5%)	0	8 (24.2%)	14 (28%)
MACULAR EDEMA	2 (12.5%)	0	10 (30.3%)	12 (24%)
MACULAR ISCHEMIA	4 (23.5%)	1	2 (6%)	7 (13.7%)

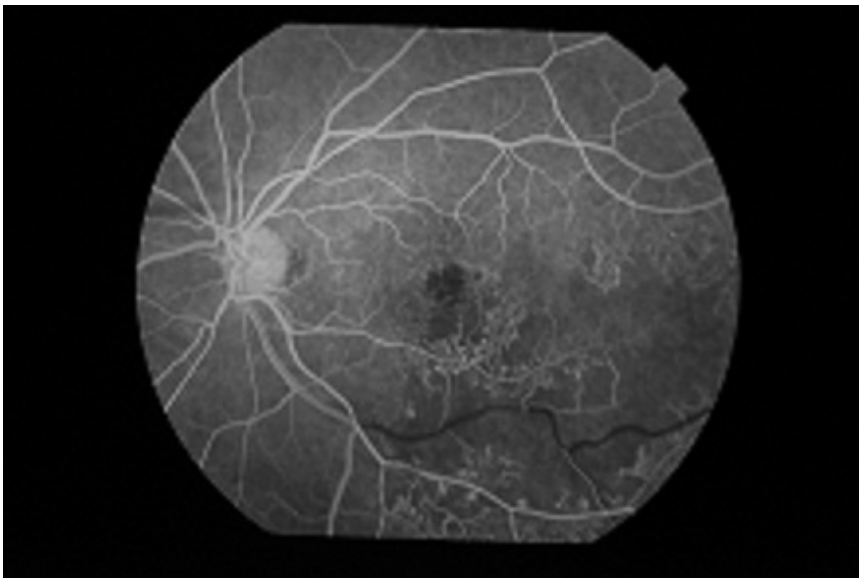
CRVO patients had more significant CNP areas, had increased incidence of neovascularisation and the presence of macular ischemia was more likely. This is because of the larger area of retina involved in CRVO


FFA FINDINGS IN CRVO
8
7
6
5
4
3
2
1
0
8
8
0
6
2
4
CNP < 10 DD
CNP > 10 DD
Collaterals
New vessels
ME
Macular ischemia

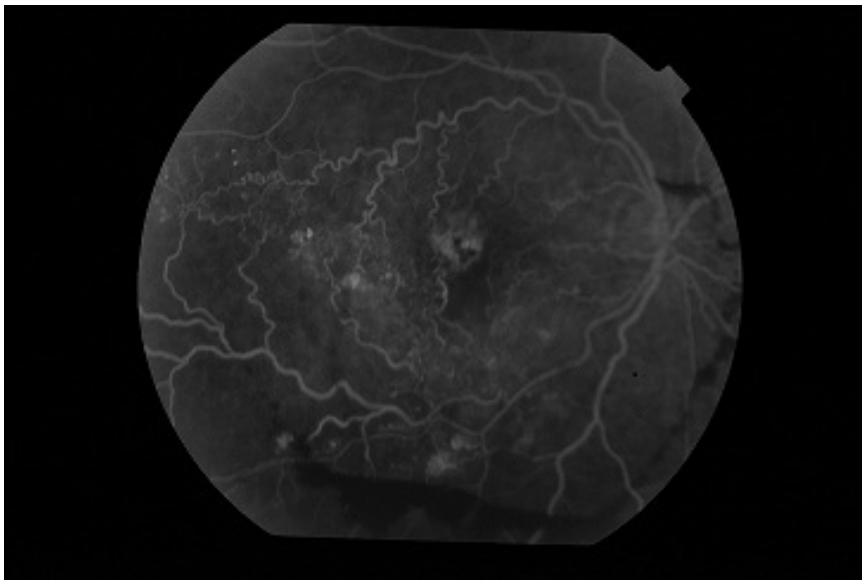

FFA FINDINGS IN BRVO
25
20
15
10
5
0
25
8
2
8
10
2
CNP < 5 DD
CNP > 5 DD
Collaterals
New vessels
ME
Macular ischemia

FFA DEMONSTRATING NON FILLING OF OCCLUDED

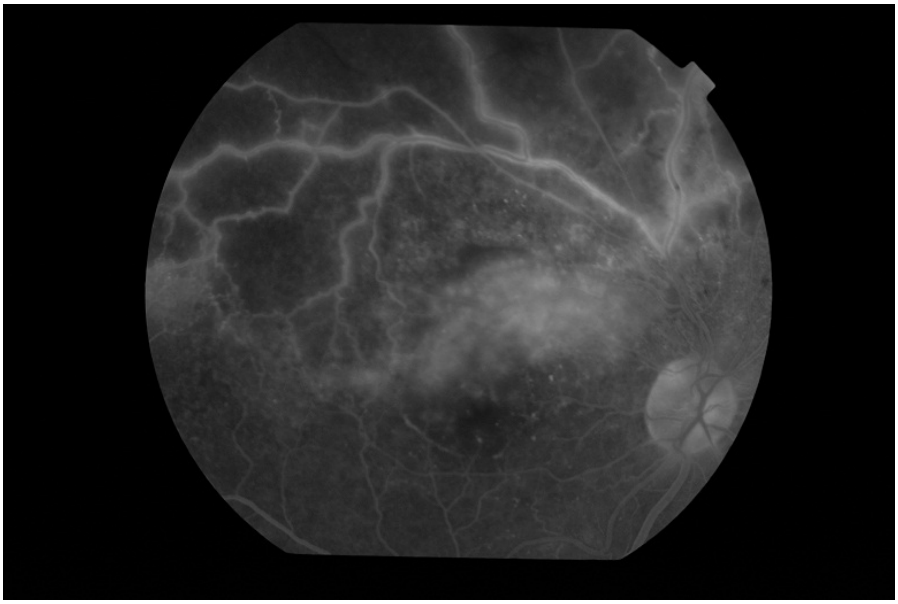
INFEROTEMPORAL VEIN WITH CAPILLARY NONPERFUSION



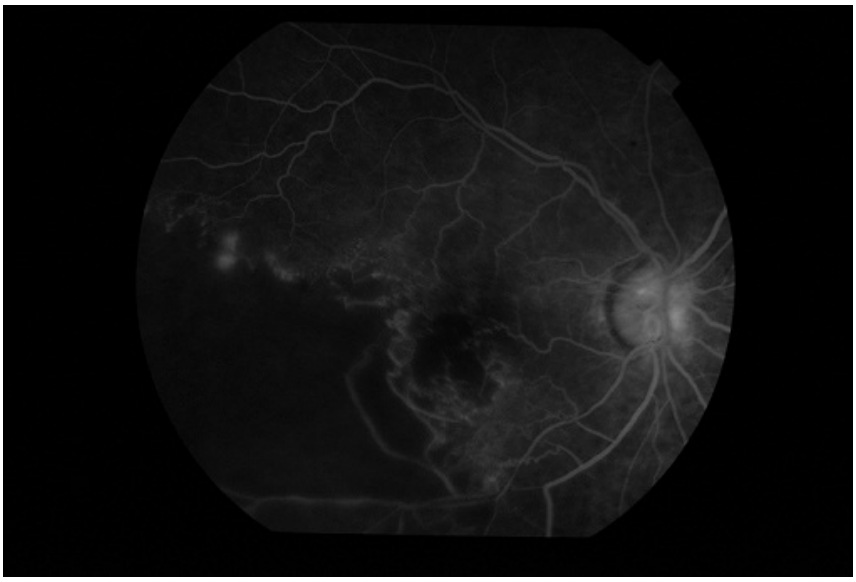
FFA SHOWING DEVELOPMENT OF COLLATERALS



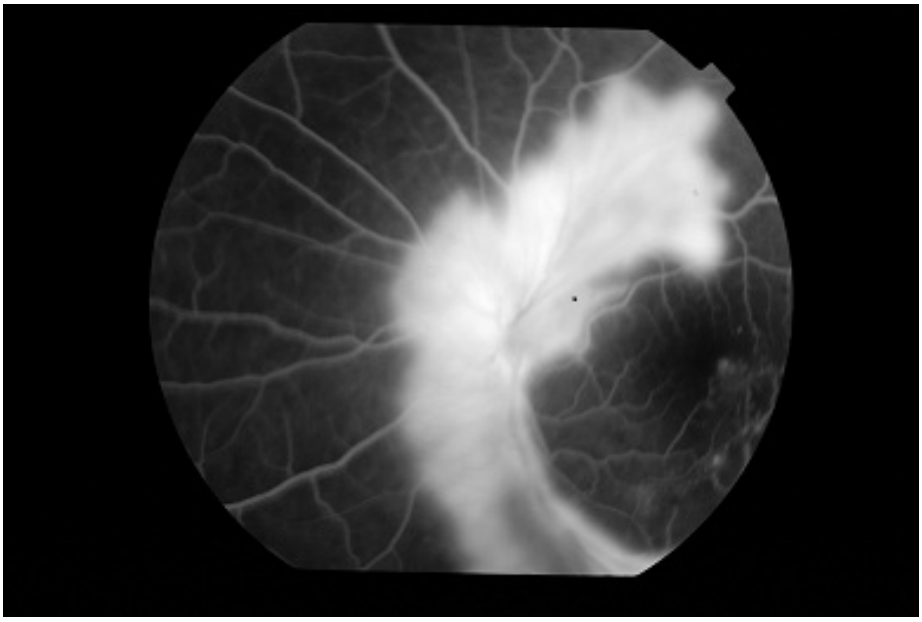
FFA SHOWING SIGNIFICANT CAPILLARY NONPERFUSION WITH MACULAR OEDEMA



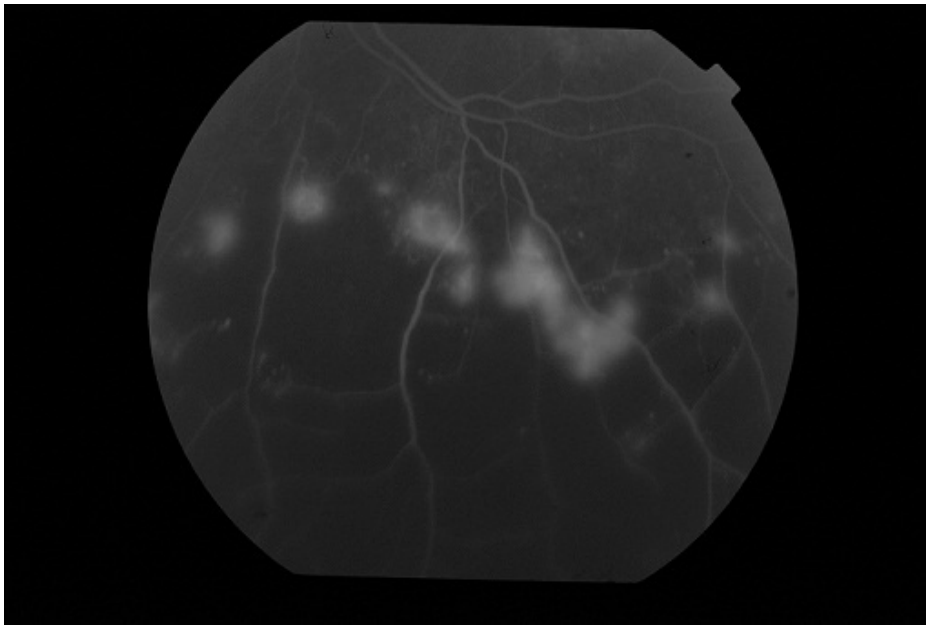
FFA DEMONSTRATING MACULAR ISCHEMIA



FFA SHOWING LEAKAGE FROM FLORID NEOVASCULARISATION



NEOVASCULARISATION ELSEWHERE



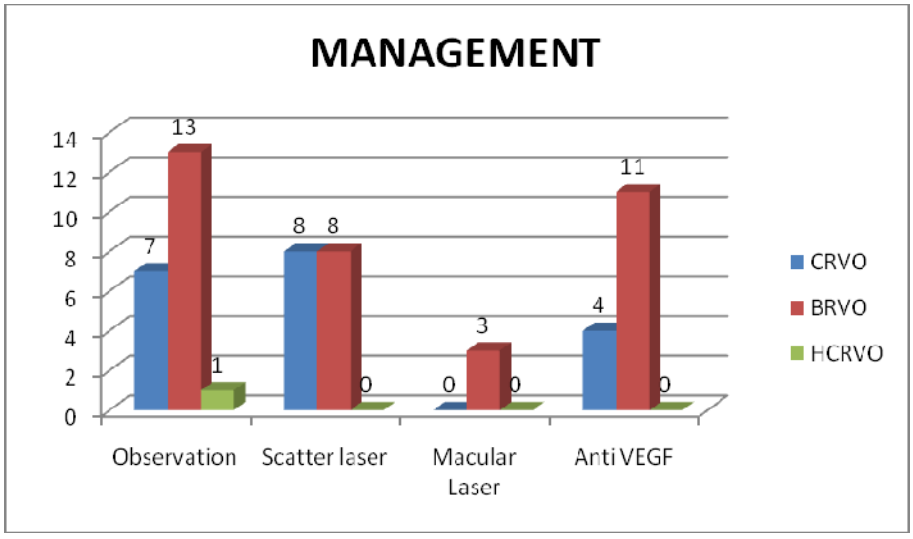
MANAGEMENT

Among the CRVO patients, 8 (47%) had either extensive capillary non perfusion area more than 10 disc diameter area or evidence of new vessels and were hence treated with panretinal scatter laser photocoagulation. In addition to laser, 2 patients were given anti-VEGF injection prior to the laser. Two patients who had macular edema were given anti-VEGF alone (1 to 2 injection). One patient of CRVO who developed non-resolving vitreous hemorrhage secondary to neovascularisation of the disc underwent pars plana vitrectomy with endolaser. Another patient had spontaneous clearing of the vitreous hemorrhage and later underwent panretinal scatter laser photocoagulation. Patients who had no evidence of new vessels or capillary non perfusion area less than 10 disc diameter area (7 patients) were observed carefully.

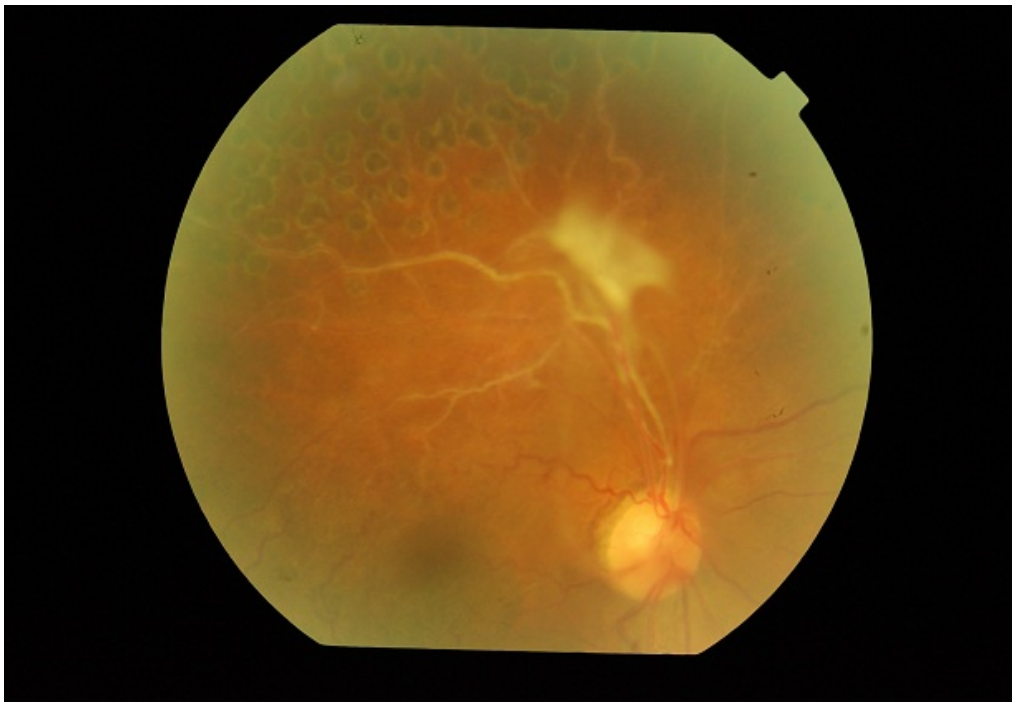
Eight (24.2%) of BRVO patients had either new vessels or capillary non perfusion areas more than 5 disc areas and were treated with sectoral laser photocoagulation. Three patients were given anti-VEGF also, in addition to laser. Nine (27.2%) patients were given anti-VEGF for macular oedema (1 to 3 injections). Macular grid laser was done in 3 patients with macular oedema due to BRVO. Observation was done in 13 (39.3%) patients who had no evidence of new vessels or capillary non perfusion area less than 5 disc diameter area. One of them with SN BRVO had a tractional detachment of the peripheral retina and hence was observed. HCRVO patient had macular ischemia and was observed.

TABLE 10 MANAGEMENT

MANAGEMENT	CRVO	HCRVO	BRVO	TOTAL
OBSERVATION	7 (41.1%)	1	13 (39.3%)	21 (41.1%)
SCATTER LASER	8 (47%)	0	8 (24.24%)	16 (31.3%)
GRID	0	0	3 (9%)	3 (5.8%)
ANTI-VEGF	4 (23.5%)	0	11 (33.3%)	15 (29.4%)



SECTORAL SCATTER PHOTOCOAGULATION

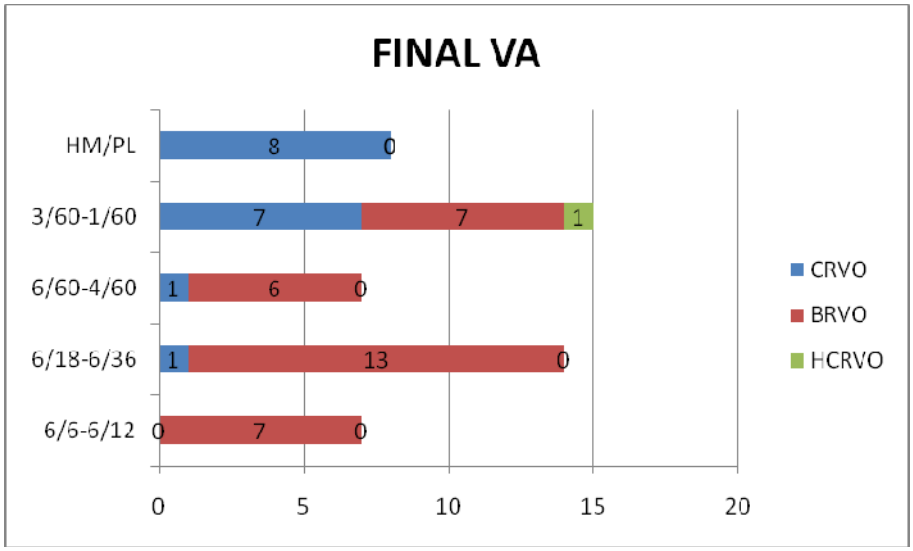


VISUAL OUTCOME

At the end of 6 months of follow-up, 15 (88.55%) patients with CRVO had final visual acuity less than 3/60 whereas only 7 (21.2%) BRVO patients had vision less than 3/60. Only 1 (5.8%) CRVO patient had vision more than 6/36 while 20 (60.6%) BRVO patients had vision more than 6/36 at the end of 6 months. The visual outcomes were much better in BRVO when compared to CRVO due to the larger area of retina involved and the higher incidence of complications in CRVO.

TABLE 11 FINAL VISUAL ACUITY

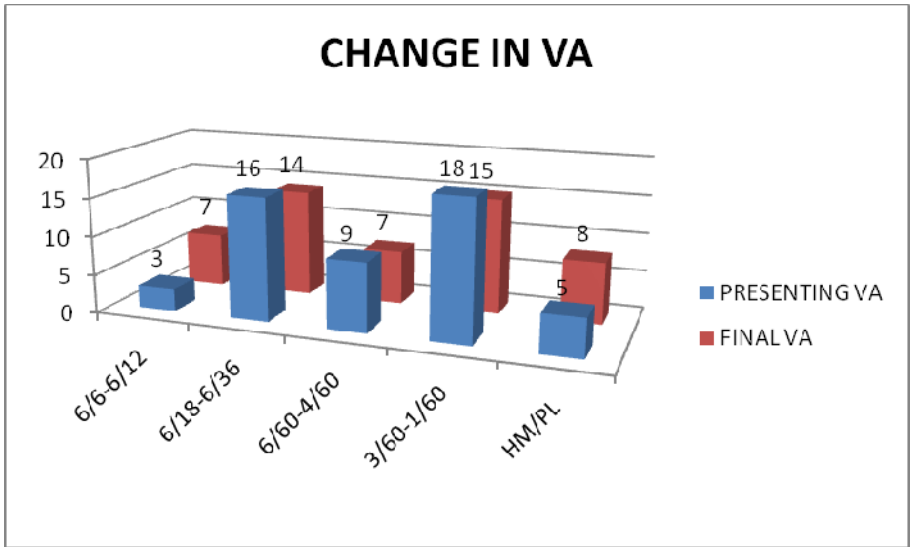
FINAL VA	**CRVO**	**HCRVO**	**BRVO**	**Total**
6/6-6/12	0	0	7	7 (13.7%)
6/18-6/36	1	0	13	14 (27.4%)
6/60-4/60	1	0	6	7 (13.7%)
3/60-1/60	7	1	7	15 (29.4%)
HM/PL	8	0	0	8 (15.6%)



CHANGE IN VISUAL ACUITY

TABLE 12 CHANGE IN VISUAL ACUITY

VISUAL ACUITY	NUMBER AT PRESENTATION	NUMBER AT 6 MONTHS
6/6 - 6/12	3	7
6/18 – 6/36	16	14
6/60 – 4/60	9	7
3/60 – 1/60	18	15
HM/ PL	5	8



At the end of 6 months of follow-up, 6 of the CRVO patients had worsening of visual acuity by 1 line, 8 had no change and 3 patients showed atleast 1 line improvement.

In BRVO, 11 patients had worsening of visual acuity, 10 had no change and 12 patients showed 1 line improvement.

The patient with HCRVO had reduction of visual acuity by one line.

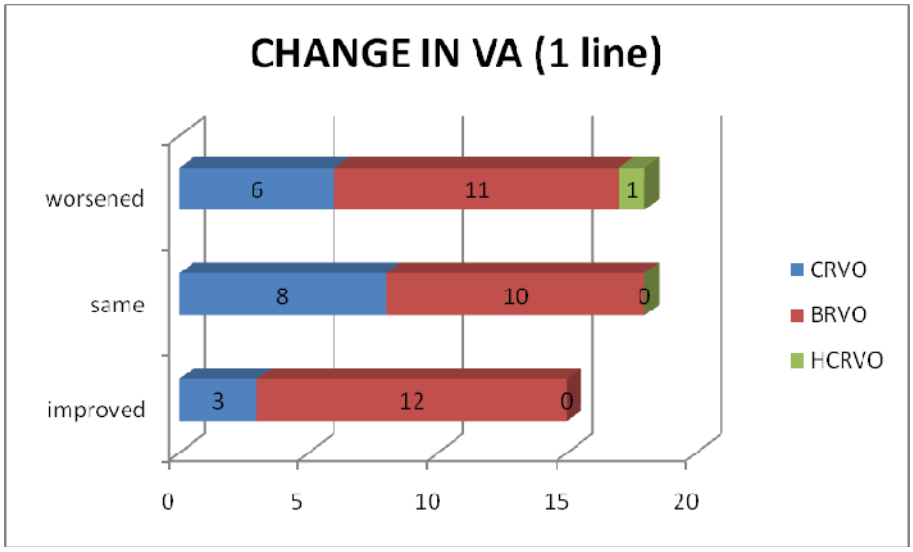
However if 2 line change in visual acuity was considered, 1 patient of CRVO had improvement by 2 lines, while the rest 16 had no change in visual acuity by more than 1 line and 5 patients of BRVO had improvement in visual acuity by 2 or more lines and the rest 28 had no change in visual acuity by more than 1 line. The HCRVO patient also did not have change in visual acuity by more than 1 line. No patient of any type had worsening of visual acuity by more than 1 line.

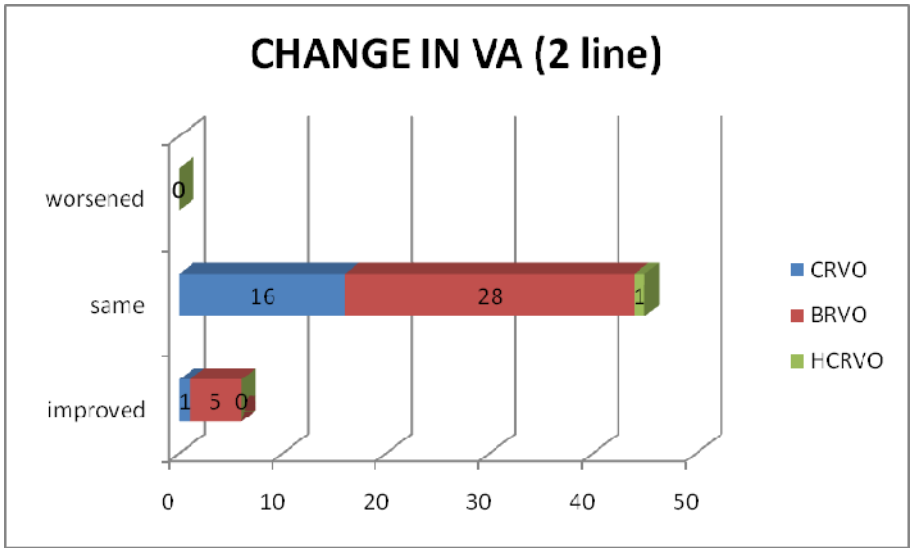
TABLE 12 CHANGE IN VISUAL ACUITY (1 line)

VISUAL ACUITY CHANGE	CRVO	HCRVO	BRVO	TOTAL
IMPROVED	3 (17.6%)	0	12 (36.3%)	15 (29.4%)
SAME	8 (47%)	0	10 (30.3%)	18 (35.2%)
WORSENED	6 (35.2%)	1	11 (33.3%)	18 (35.2%)

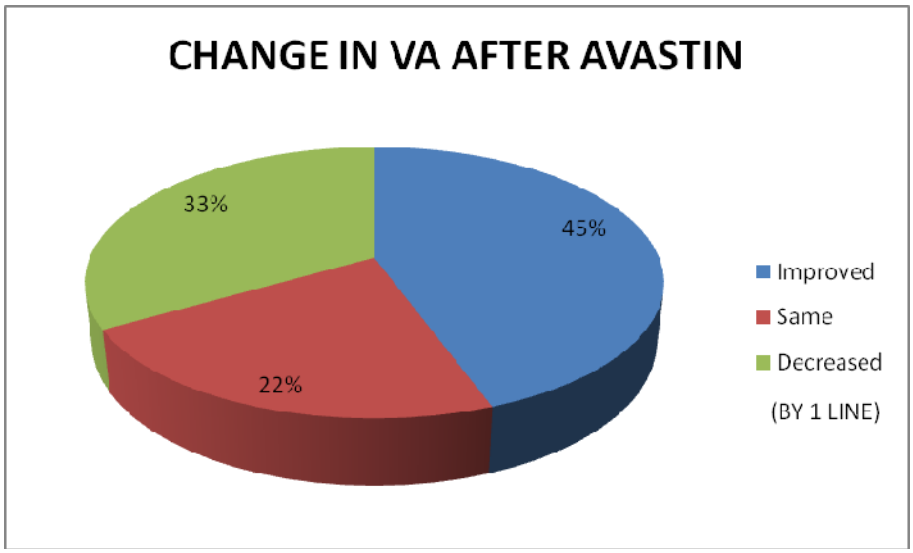
TABLE 13 CHANGE IN VISUAL ACUITY (2 line)

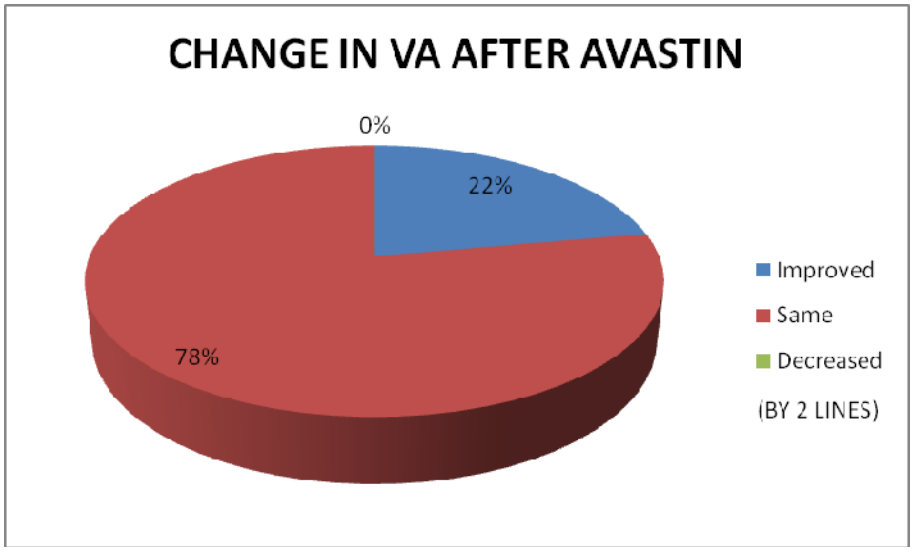
VISUAL ACUITY CHANGE	CRVO	HCRVO	BRVO	TOTAL
IMPROVED	1 (5.9%)	0	5 (15.2%)	6 (11.8%)
SAME	16 (94.1%)	1	28 (84.8%)	45 (88.2%)
WORSENED	0 (0%)	0	0 (0%)	0 (0%)





None of the patient with macular oedema who were treated with anti-VEGF injection or macular grid lost more than 1 line, while 2 patients gained 2 lines and one patient had a dramatic 4 line improvement in vision.

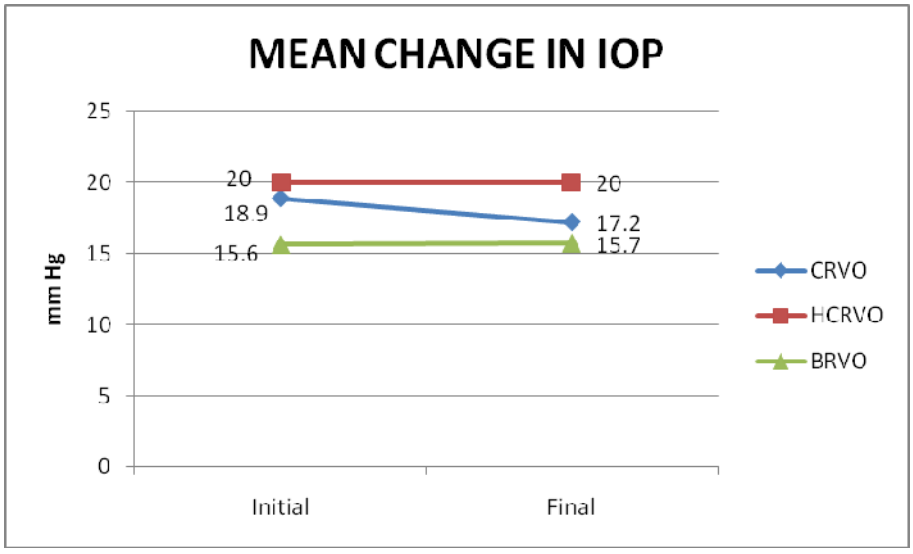




Taking the eyes which received Avastin for macular oedema alone, 67% had either same or improvement in vision by atleast 1 line and 33% had decrease in vision by 1 line alone. None of the patients had drop in vision of more than 1 line and 27% had improvement in vision by atleast 2 lines.

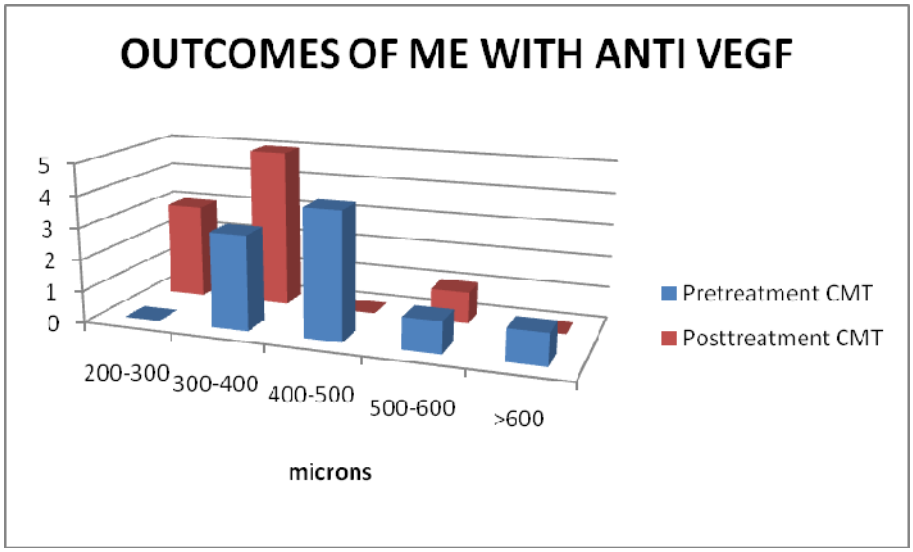
CHANGE IN INTRAOCULAR PRESSURE

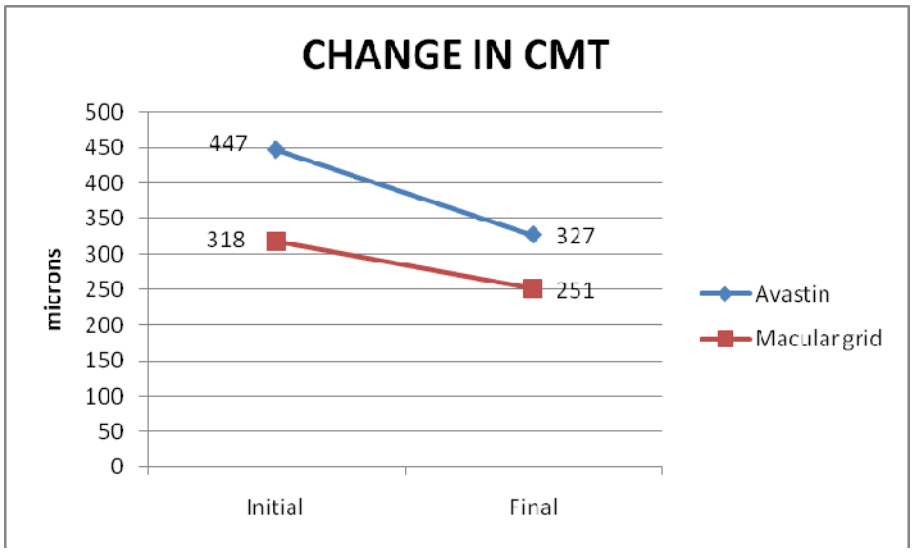
The mean intraocular pressure did not change much from 16.8 ± 5.4 mm Hg (ranging from 10 to 48 mm Hg) at presentation to 16.3 ± 2.9 mm Hg (ranging from 12 to 28 mm Hg). The patients with primary open angle glaucoma and neovascular glaucoma were under medical management. Also none of the patients were treated with steroids.



The mean IOP in Avastin group was 17.6mm Hg before treatment and 16 mm Hg after treatment.

CHANGE IN CENTRAL MACULAR THICKNESS

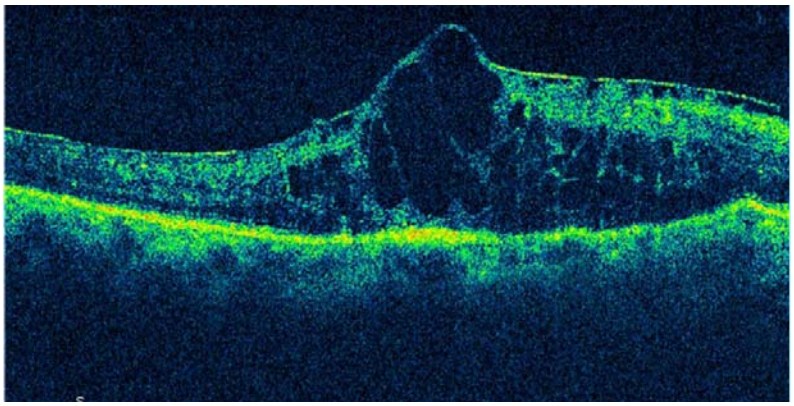




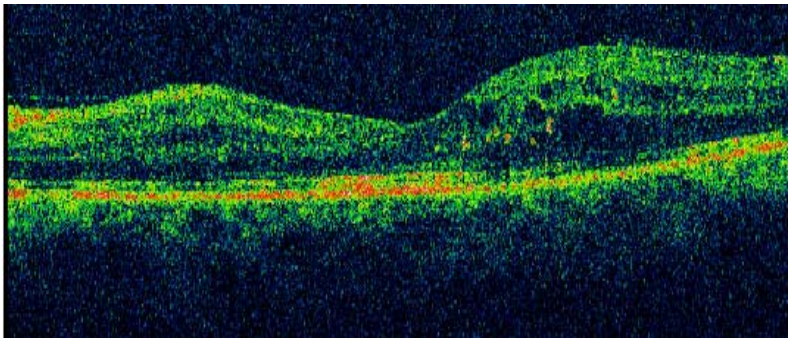
In the 9 patients with macular oedema, who were treated with intravitreal anti-VEGF (Avastin) injection, the mean central macular thickness changed from 447µ to 327µ. Prior to treatment, 66.6% had central macular thickness of more than 400µ. After treatment, 88.8% of the patients had central macular thickness of less than 400µ .

The mean central macula thickness also reduced form 318µ to 251µ in the 3 patients who underwent macular grid laser for macular oedema.

OCT SHOWING GROSS MACULAR OEDEMA IN CRVO



OCT SHOWING SEGMENTAL MACULAR OEDEMA IN BRVO



SUMMARY

1. Fifty one eyes of 51 patients with retinal vein occlusion were studied.

2. Out of the 51 cases, 17 were CRVO, 33 were BRVO and one patient had HRVO. CRVO constituted 33%, BRVO constituted 65% and HRVO was 2%.

3. Most of the patients were in their 6^{th} and 7^{th} decades with a mean age of 56.2 years.

4. Male preponderance was noted. Overall 67% were male. This could be due to increased incidence of smoking in males.

5. Of the 33 patients with BRVO studied, 20 had ST BRVO, 10 had IT BRVO, 1 had SN BRVO and 2 had macular BRVO.

6. The commonest comorbid condition was hypertension, which was found in 28 (53%) patients. Diabetes mellitus was present in 10 (19.6%) patients. Four patients had cardiac abnormalities, 2 had cerebrovascular accident and 4 had POAG.

7. Among CRVO cases, 3 (17.6%) had diabetes mellitus, 9 (52.9%) were hypertensive, 8 (47%) had hypercholesterolemia, 1 (5.8%) had a history of CVA and 2 (11.7%) had POAG.

8. Among the BRVO patients, 7 (21.2%) were diabetic, 18 (54.9%) were hypertensive. 14 (42.4%) had hypercholestolemia, 4 (12.1%) had CAD, 1(3%) had CVA and 2 (6%) had POAG.

9. Of the CRVO patients, 5 (29.4%) presented within 4 weeks of the onset of venous occlusion, while 6 (18.1%) BRVO patients presented within 4 weeks. The 3 CRVO patients who developed neovascular glaucoma all presented after 20 weeks.

10. Eight (50%) CRVO patients had CNP areas more than 10 disc diameter area, 6 (37.5%) had new vessels, and 2 (12.5%) had macular oedema. Macular ischemia was noted in 4 (25%) of patients with CRVO.

11. Of the BRVO patients, 8 (24.2%) had CNP more than 5 disc diameter area, 8 (24.2%) had new vessels, 10 (30.3%) had macular oedema and 2 (6%) had macular iscemia.

12. If 2 line change in visual acuity was considered, 1 patient of CRVO had improvement by 2 lines, while the rest 16 had no change in visual acuity by more than 1 line . 5 patients of BRVO had improvement in visual acuity by 2 or more lines and the rest 28 had no change in visual acuity by more than 1 line. The HCRVO patient also did not have change in visual acuity by more than 1 line. No patient of any type had worsening of visual acuity by more than 1 line.

13. Among the eyes which received Avastin for macular oedema, 67% had either same or improvement in vision by atleast 1 line and 33% had decrease in vision by 1 line alone. None of the patients had drop in vision of more than 1 line and 27% had improvement in vision by atleast 2 lines.

14. In patients with macular oedema, treatment with Anti-VEGF injection or macular grid laser reduced the central macular thickness.

15. There was no significant change in the mean intraocular pressure on followup.

CONCLUSIONS

- Retinal venous occlusions are common in the 6^{th} and 7^{th} decades
- It is seen more commonly in males.
- The most common predisposing factor for retinal vein occlusion was hyertension.
- Branch retinal vein occlusions were commoner than central retinal vein occlusions and had better visual outcomes.
- All the patients had either same visual acuity or had improvement in visual acuity by 2 or more lines. None of them had worsening of visual acuity by more than 1 line.
- In our study, 9 patients with macular edema were treated with anti-VEGF and 3 were treated with grid laser. In both these groups, the mean central macular thickness was reduced after treatment.
- Finally we conclude that
    - In CRVO, final vision is limited despite available treatment. However, regular monitoring is required to detect and treat complications.
    - In BRVO, there is chance for visual improvement in patient with initial good vision.

PROFORMA

A CLINICAL STUDY ON RETINAL VASCULAR OCCLUSIVE DISORDERS

Name		Age	Gender	OP/IP No
Address				Date

Presenting Complaints

Defective Vision	Yes/No	OD/OS	Duration	Sudden/Gradual
Field Loss	Yes/No	Quadrant		

Floaters/Flashes/Macropsia/Micropsia/Metamorphopsia/Scotoma

Any other symptoms

Past History

Similar illness

Glaucoma Drug history

HT/DM/Hyperlipidemia/CVS diseases/Blood dyscrasias/Infections Duration

Personal History

Smoking/Alcohol

Family History

Ocular Examination

BCVA	OD	OS
IOP		
RAPD		

Anterior segment examination

(NVI/NVA)

Fundus examination

Investigations

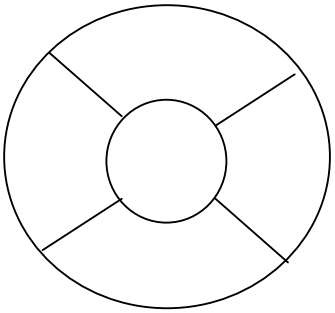
FFA

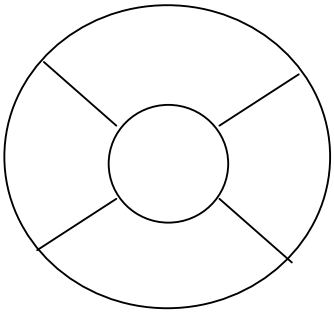
Site of occlusion/CNP/Collaterals/New vessels

Macular oedema/ischemia

Disc leakage/Staining of vessel walls

OCT





Visual Fields

BP

RBS	Hb	TC	DC	ESR
Peripheral smear	Platelets		BT	CT
Blood urea	Creatinine			
Lipid profile				
Urine albumin/sugar				
VDRL	Mantoux		CXR	
Cardiology opinion				
Septic foci-ENT/Dental/Skin/VD/Gynaec				

Treatment given

Follow up

	4 weeks	**12 weeks**	**24 weeks**
BCVA			
IOP			
Anterior segment			
Fundus			
OCT			

	OP No	Name	Age	Sex	Eye	Diagnosis	DV duration(weeks)	Risk Factors	RBS	BP	S cholesterol	Initial VA	Initial IOP	FFA findings	Initial CMT	Treatment	No of Avastins	Final VA	Final IOP	Final CMT	Complications
1	69989	CHANDRA	50	F	OS	ST BRVO	1	HT	80	200/100	227	6/12	16	CNP		Observation		6/12	14		
2	468934	JANAKIRAMAN	61	M	OS	IT BRVO	24	POAG	96	120/86	210	3/60	14	CNP		Observation		3/60	16		ERM
3	78929	ELUMALAI	44	M	OS	IT BRVO	8	Nil	102	112/78	192	6/18	14	CNP		Observation		6/24	12		
4	857	RAJAN	54	M	OD	CRVO	1	CVA/DM	120	140/80	230	1/60	18	ISCHEMIA		Observation		1/60	18		
5	4732	DAMODARAM	76	M	OS	ST BRVO	4	HT/CAD	94	170/110	168	3/60	10	CME	468	Avastin	3	6/36	16	276	ME
6	396	PREMKUMAR	53	M	OD	CRVO	1	HT	90	150/100	200	1/60	14	ISCHEMIA		Observation		HM	12		
7	4826	PONNI	50	F	OS	ST BRVO	1	HT	120	160/100	250	6/60	18	CME	397	Avastin	2	6/24	14	337	ME
8	12047	VENKIAH	58	M	OD	CRVO	22	HT	130	170/100	170	1/60	48	NVD/NVE/CNP		PRP/avastin		HM	28		NVG
9	759	VISWANATHAN	51	M	OD	CRVO	1	POAG	100	110/80	205	1/60	22	CNP		Observation/AGM		1/60	18		
10	6548	KANNIAPPAN	51	M	OS	Mac BRVO	8	HT	94	160/90	161	6/18	16	CME	321	Avastin	1	6/12	12	267	ME
11	15694	MARIAMMAL	65	F	OS	IT BRVO	12	HT	125	170/90	211	6/60	20	CNP		Observation		6/60	18		
12	14824	GANDINESAN	47	M	OS	IT BRVO	4	Nil	134	150/90	242	6/18	18	CNP		Observation		6/12	18		
13	4146	DASARATH	60	F	OS	CRVO	4	HT	79	150/100	152	HM	18	ME	544	Avastin	2	HM	16	323	ME
14	7215	RENUKA	65	F	OD	IT BRVO	12	DM/HT	184	170/100	230	6/12	18	NVE		Avastin/PRP	1	6/9	20		
15	64151	MANNAN	40	M	OS	ST BRVO	24	HT/CAD/CVA	100	130/80	173	6/24	12	ME	412	AVASTIN	2	6/36	12	223	ME
16	10140	MUTHUKRISHNAN	73	M	OD	ST BRVO	24	Nil	180	130/90	201	1/60	20	CNP/ISCHEMIA		PRP		1/60	16		
17	15380	JON BEGUM	65	F	OD	ST BRVO	20	HT	98	170/100	221	6/24	14	NVE		PRP		6/36	16		
18	15405	SERMATHAYEE	58	F	OD	ST BRVO	24	HT/DM	230	140/100	187	6/18	18	NVD/CNP		PRP		6/24	18		
19	59486	GOVINDAMMAL	38	F	OS	CRVO	12	Nil	167	120/80	225	6/24	14	CNP		Observation		6/36	16		
20	479264	KUMAR	55	M	OS	CRVO	36	Nil	130	140/90	169	HM	14	NVE/CNP		Avastin/PRP	1	HM	14		NVG
21	13706	RATHINAM	53	F	OD	CRVO	16	HT	152	160/100	146	3/60	18	Not done due to VH		Observation		2/60	20		VH
22	20510	DANAPAL	69	M	OD	ST BRVO	14	DM/HT	180	170/90	247	1/60	12	ME	446	Avastin	2	1/60	14	302	ME
23	3405	PALANI	75	M	OS	ST BRVO	12	DM/HT	165	170/90	124	6/36	12	ME	412	Avastin	2	6/24	12	301	ME
24	472238	POONGAVANAM	60	F	OS	IT BRVO	36	Nil	121	140/90	157	1/60	14	ME	396	GRID		3/60	14	243	ME
25	42769	WILSENT	58	M	OD	ST BRVO	8	HT	160	150/100	189	6/18	12	CNP		Observation		6/12	14		
26	7194	PALANI	35	M	OS	ST BRVO	24	HT/CAD	143	140/90	165	6/24	18	NVD/CNP		Avastin/PRP	1	6/18	16		
27	479869	DARMAN	65	M	OS	CRVO	32	POAG	154	130/90	202	2/60	18	NVD		PPV		1/60	20		VH
28	88563	RADHAKRISHNAN	59	M	OD	ST BRVO	3	DM/HT	210	150/110	162	6/24	12	NVE/NVD		PRP		6/18	16		
29	67262	PERIAMMAL	66	F	OD	CRVO	4	Nil	136	110/74	192	PL	16	CNP		Observation		PL	14		
30	92160	ANTHONY	38	F	OS	SN BRVO	4	DM/HT	186	146/94	171	6/9	18	COLLATERALS		Observation		6/9	16		peripheral TRD
31	53757	RANI	60	F	OD	CRVO	12	Nil	90	140/80	173	PL	14	CNP/ISCHEMIA		PRP		PL	14		
32	12337	SRINIVASAN	65	M	OD	CRVO	20	HT	105	140/90	264	2/60	20	NVE		PRP		2/60	14		NVG
33	10958	KUMAR	39	M	OD	ST BRVO	16	Nil	88	104/68	211	6/18	20	NVE		PRP		6/18	14		
34	74481	SEKAR	44	M	OD	ST BRVO	22	DM	156	132/88	198	4/60	12	CNP		Observation		6/60	18		
35	50450	MANAVALAN	72	M	OS	ST BRVO	18	Nil	97	114/72	231	4/60	16	ISCHEMIA		Avastin		3/60	16		
36	4374	SIVADAS	55	M	OS	Mac BRVO	26	Nil	142	132/84	165	6/18	22	COLLATERALS		Observation		6/9	18		
37	58383	CHANDRAN	61	M	OD	CRVO	14	HT	139	140/100	210	HM	18	CNP NVD		PRP		HM	20		
38	16284	MARIMUTHU	61	M	OS	HCRVO	1	HT	139	140/100	210	3/60	20	COLLATERALS/ISCHEMIA		Observation		2/60	20		
39	4259	NAGABUSHANAM	35	M	OD	CRVO	8	Nil	142	140/90	163	1/60	14	CNP		PRP		2/60	18		
40	42405	VENU	60	M	OD	CRVO	12	HT	112	150/100	152	2/60	20	NVD		PRP		4/60	16		
41	21011	YUVARAJ	40	M	OD	ST BRVO	14	Nil	136	140/80	189	6/24	18	CNP		Observation		6/36	20		
42	8044	HARIKRISHNAN	42	M	OS	IT BRVO	6	Nil	110	130/80	212	6/36	14	CNP		Observation		6/36	14		
43	55567	MUTHUMARI	75	M	OD	ST BRVO	16	Nil	176	150/90	190	5/60	12	CNP/ME	235	GRID		4/60	14	243	ME
44	67973	ANJALAI	60	F	OD	CRVO	10	HT/DM	219	150/100	187	1/60	16	ISCHEMIA		Observation		HM	18		
45	71201	KANNIAMMAL	64	F	OD	IT BRVO	20	HT	149	160/80	165	3/60	18	ME	323	GRID		2/60	18	267	ME
46	407112	ANTONY	70	M	OD	ST BRVO	18	POAG	98	130/80	264	4/60	16	CNP		Observation/AGM		4/60	14		
47	10201	DASARATHAN	62	M	OD	ST BRVO	30	Nil	105	140/90	183	6/60	14	NVE		PRP/ Avastin	1	6/60	16		
48	76411	SELVARAJ	60	M	OS	IT BRVO	8	HT/CAD	164	200/110	144	2/60	18	ME	356	Avastin	2	1/60	18	369	ME
49	56120	RANJITHAM	63	F	OD	ST BRVO	16	HT	135	130/80	190	6/60	16	CNP		Observation		6/36	18		
50	90555	RAMACHANDRAN	55	M	OS	CRVO	12	HT/DM	111	128/74	256	4/60	20	ME	642	Avastin	1	3/60	16	544	ME
51	69874	MALAR	35	F	OD	IT BRVO	24	Nil	105	126/76	211	6/36	12	NVE		PRP		6/60	16		VH

ABBREVIATIONS

1. M-male
2. F-female
3. OD-Right eye
4. OS- Left eye
5. HT-hypertension
6. DM-diabetes mellitus
7. CAD-coronary artery disease
8. CVA-cerebrovascular accident
9. ST-superotemporal
10. SN-superonasal
11. IT-inferotemporal
12. IN-inferonasal
13. VH-vitereous haemorrhage
14. ME-macular edema
15. CME-chronic macular edema
16. NVD-neovascularisation disc
17. NVE-neovascularisation elsewhere
18. CNP-capillary non-perfusion
19. PRP-pan retinal photocoagulation
20. PPV-pars plana viterectomy
21. NVG-neovascular glaucoma
22. TRD-tractional retinal detatchment
23. AGM-anti glaucoma medication
24. ERM-epiretinal membrane
25. CRVO-central retinal vein occlusion
26. BRVO-branch retinal vein occlusion

27. AV-arterio-venous
28. VA-visual acuity
29. RAPD-relative afferent papillary defect
30. ERG-electroretinogram
31. FFA-fundus fluorescein angiography

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